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BRAIN.

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THE STRUCTURAL BASIS OF TRAUMATIC EPILEPSY AND RESULTS OF RADICAL OPERATION.[1]

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EPILEPSY is a specific reaction on the part of the central nervous system to a noxious stimulus. It may be called focal when it is possible to determine a circumscribed area upon which the stimulus first acts. Thanks to recent advances in neurophysiology and in neurosurgery it is now possible to include in the group of focal epilepsy many cases formerly considered to be generalized. In addition to the march of crude motor phenomena characteristic of Jacksonian epilepsy, alterations in the directions of gaze, turning of the head and certain sensory auras may serve to point to the focus from which an epileptogenic stimulus is derived.

Even in traumatic epilepsy, however, where the irritative focus may be easily localized there are other factors to be considered. In addition to the irritative stimulus the occurrence of a convulsion depends upon the irritability and the susceptibility of the brain to such stimulus. Further, there are certain general influences which though they operate from a distance directly influence the production of convulsions. There can be little doubt but that epilepsy is an active stimulation process and not a release phenomenon, although release phenomena may be observed during and following an attack.

Exploration of the human cerebral cortex, both in normal and

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pathological areas in well over 100 cases under local anæthesia, has made it possible to outline certain definite epileptogenic cortical areas (Foerster [4]). The areas in the human cortex are analogous to but not identical with the areas outlined by Vogt in monkeys. The movement patterns which follow such stimulation experiments provide a local sign which often makes it possible to localize the irritating focus of an epileptic discharge, even where there are no physical signs to suggest this localization.

Quantitative stimulation in the post-central field (3, 1, 2) (fig. 1)

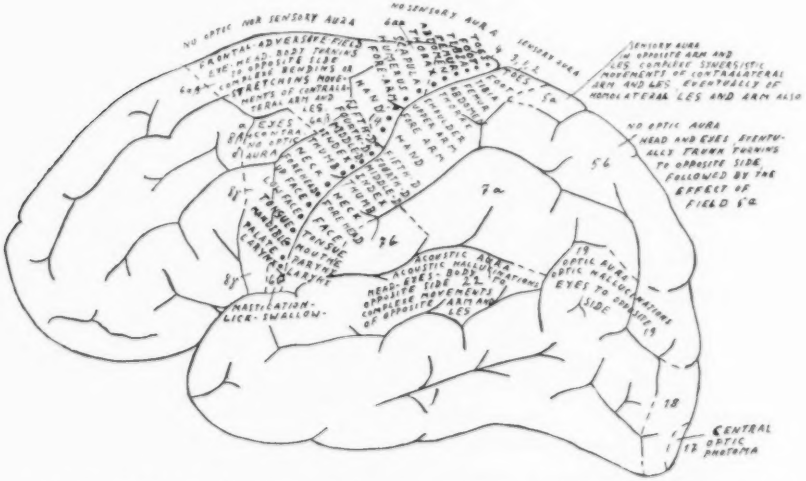
NO OPTIC NOR SENSORY AURA
NO SENSORY AURA
SENSORY AURA
SENSORY AURA IN OPPOSITE ARM AND LEG COMPLEX SYNERGISTIC MOVEMENTS OF CONTRALATERAL ARM AND LEG EVENTUALLY OF HOMOLATERAL LEG AND ARM ALSO
NO OPTIC AURA HEAD AND EYES EVENTUALLY TRUNK TURNING TO OPPOSITE SIDE FOLLOWED BY THE EFFECT OF FIELD 6α
FRONTAL ADVERSIVE FIELD EYES, HEAD, BODY TURNING TO OPPOSITE SIDE CONVULSIVE BENDING OR STRETCHING MOVEMENTS OF CONTRALATERAL ARM AND LEG
EYES CONTRA NO OPTIC AURA
TRUNK
HAND
FOREARM
UPPER ARM
SHOULDER
HIP
KNEE
FOOT
TOES
LEG
NECK
FACE
HEAD
TRANSVERSE FACIAL
MOUTH
PHARYNX
LARYNX
MASTICATION-LICK-SWALLOW
ACOUSTIC AURA ACOUSTIC HALLUCINATIONS
HEAD-EYES-BODY TO OPPOSITE SIDE COMPLEX MOVEMENTS OF OPPOSITE ARM AND LEG
OPTIC AURA OPTIC HALLUCINATIONS EYES TO OPPOSITE SIDE
CENTRAL OPTIC AMAUROSIS

FIG. 1.—Epileptogenic cortical areas in the human brain. The outlines indicate roughly the initial movements and auræ which follow direct stimulation of the cortex by an electrical current sufficient to produce an epileptic seizure.

requires a considerably larger amount of current to bring on an attack than in the pre-central (4 + 6a *α*). The clonic convulsion produced by stimulation in either area appears to be about the same. If it arises, however, behind the central fissure the attack is preceded by a sensory aura which begins in the part first affected and often spreads like a wave over that side of the body.

Stimulation in the frontal adversive field (6a *β*) (fig. 1) produces turning of eyes, head and body to the opposite side, followed by clonic or tonic movements of the contralateral arm and leg. These movements take place without any aura. Below this field the frontal eye field may give rise to a convulsion initiated by isolated clonic turning of the

eyes to the opposite side without any optic aura. Following this the attack takes on either the characteristics of a frontal adversive field attack, or a simple pre-central attack, depending upon the direction in which the stimulus spreads.

Stimulation of the occipital pole in area 17 produces an attack which is ushered in by an optic aura such as light, flames, stars, &c., usually in the opposite visual field. Stimulation farther forward (area 19) causes a change in the direction of the gaze to the opposite side associated with an optic hallucination, such as a figure in the opposite visual field. A stimulus in the parietal adversive field (5b + 5a) produces turning of the eyes, head and body to the opposite side and movements of the opposite limbs. If it be in the anterior portion of this area (5a) the movements of the head and eyes may be absent, but there is a sensory aura of discomfort in the arm and leg of the opposite side followed by leg and arm movements. As indicated in fig. 1, a stimulus in the posterior temporal region is likely to produce an acoustic aura. Lesions affecting the operculum centrale (area 6b) cause grunting, licking, swallowing or some such movement of the mouth and pharynx, associated usually with a sensation in this vicinity and also singultus.

In any case of focal epilepsy where there is a reasonable suspicion of a cerebral lesion, *encephalography* should be employed to demonstrate any gross cerebral abnormality. If operation be then undertaken a careful electrical exploration of the cortex under local anæsthesia serves to prove whether or not the suspected focus is actually the site of the initiation of the discharge.

The radical excision of a lesion, even though it is obviously the focus for epileptic discharges, can only be a rational procedure if the lesion produced by the excision be a more innocent one than the original focus. A comparison of the results of cerebral laceration and excision has been made the subject of a series of experiments by Penfield [1] which indicate a striking difference in the end-result of excision from that of laceration.

If a wound be made in the brain and the damaged cerebral tissue is not removed there forms rapidly a scar which contains connective tissue and fibrous astrocytes, and which is densely attached to the overlying meninges. A very rich plexus of new-formed vessels appears in the scar. This plexus anastomoses on the one hand with the extra-cerebral blood supply of meninges and scalp, and on the other hand with the vessels of the brain. The cicatrix contracts steadily over a period of years. There results a very considerable pull upon the vaso-astral framework of the

brain. This framework is made up of the blood-vessels and attached fibrous astrocytes. The pull exerts an influence on the whole hemisphere and, in fact, upon the whole brain.

The effect of such a pull after a brain injury has been pointed out by Foerster [2]. He noted that there was a "wandering" of both lateral and third ventricles towards the site of such a lesion. The cause of this phenomenon is now apparent, as will be pointed out below. The displacement is due to cicatricial *pull* probably associated with the atrophy which takes place in the brain about the cicatrix.

Excision of cerebral tissue, however, produces a different result. There is no organization of connective tissue. Instead of that there appears a fluid-filled space with a very small amount of gliosis about it, without a plexus of new-formed vessels and without evidence of cicatricial pull upon the surrounding brain. Thus the results of brain injury and brain excision are structurally quite different.

Nevertheless, in the last analysis of course, the wisdom of substituting a non-contracting fluid-filled space for a contracting cicatrix in these patients must be judged on a human empirical basis.

The cases to be reported below provide an opportunity for studying the result of the excision of cerebral cicatrix in epileptics and the mechanism involved. In the group of cases selected the gunshot wounds of the brain are of long duration and the birth injuries of still longer. Consequently the excised tissue offers a remarkable opportunity to study the late results of cerebral trauma histologically.

Case Reports.

Case 1.—In 1914 the patient received a gunshot wound in the left parietal region. This was followed by twelve hours of unconsciousness and a hemiparesis and hemi-ataxia of the extremities of the right side. Ataxia and intention tremor have persisted in the right arm together with loss of cortical sensibility. The patient learned to write with his left hand. The onset of Jacksonian epilepsy involving the right side began in 1920, i.e., six years after the trauma.

Encephalography in 1925 showed some enlargement of the left lateral ventricle and a displacement of both lateral and third ventricles towards the side of the lesion (fig. 2).

Operation, 1925.—A large parietal craniotomy was carried out and dura mater was found to be densely adherent to an underlying brain scar. Within the scar was a large cyst which did not communicate with the ventricles. Electrical exploration showed the lesion to lie just posterior to the motor area for the fingers. A wide excision was carried out as indicated by the broken line in

fig. 3. The bone flap was not replaced. The patient made a good recovery and in the five years following the operation up to the time when last heard of he was quite free from convulsive seizures.

FIG. 2.—*Case* 1. Encephalogram (brow up) eleven years after gunshot wound of left parietal region. Cranial defect is shown and lateral ventricles pulled towards it.

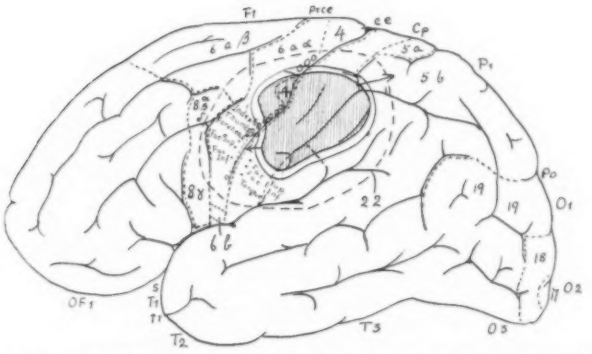

FIG. 3.—*Case* 1. Shaded area shows the position of the lesion at operation and the second line the extent of operative removal. The pattern of the attacks indicated that the stimulus might arise in front of or behind the lesion, as indicated by the arrows.

The gross specimen showed considerable thickening of the dura. A bit of bone was embedded in the cicatrix and bands of fibrous tissue could be seen passing downwards into the brain (fig. 4).

Microscopical examination shows the connective tissue to be entering the brain scar in more or less parallel lines (fig. 5). Closer examination shows these bands to be made up of blood-vessels and sturdy collagen fibres. When stained so as to demonstrate both collagen fibres and neuroglia fibres, it is seen that the two types of fibres are more or less intermingled and parallel as though they were strands in a rope. Deeper in the brain neuroglia fibres are found

FIG. 4.—*Case* 1. Cross section of cicatrix attached to thickened dura.

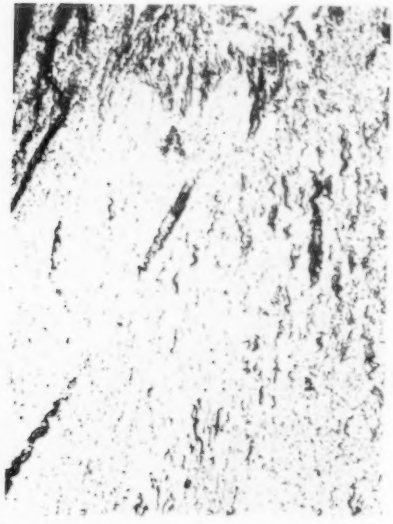

FIG. 5.—*Case* 1. Connective tissue and vessels entering the brain scar in parallel strands. Silver carbonate collagen stain.

lying in parallel lines, pointing upwards towards the meningo-cerebral adhesion (fig. 6).

In one sense this tissue can no longer be called cerebral tissue inasmuch as it no longer contains either nerve-cells or nerve-fibres. A very occasional nerve-fibre may be found in these areas of gliosis.

It seems obvious from the structure of this tissue that it is quite capable of transmitting the pull which the contracting cicatrix must have exerted

FIG. 6.—*Case* 1. Parallel neuroglia fibres oriented in the direction of the cicatricial pull. Silver carbonate astrocyte stain, Hortega.

FIG. 7.—*Case* 2. Encephalogram (brow up), fourteen years after gunshot wound of right parietal region; shows cranial defect, the underlying brain cyst, enlargement of right lateral ventricle and pulling of all three ventricles in the direction of the defect.

upon the brain itself, as evidenced by the migration of the ventricles towards the side of the lesion.

Case 2.—The patient received a gunshot wound in the right parietal region in 1914, which was followed by hemiplegia of the extremities on the left side. The hemiplegia, which has only partially disappeared was followed in 1919 by Jacksonian epilepsy of parietal field type (see legend with fig. 8).

Encephalography in 1928 showed the right lateral ventricle to be considerably enlarged. The air entered a cyst just beneath the cranial defect which was due to the wound. Both lateral ventricles and the third ventricle were deviated across to the right side and the right ventricle upward toward the defect (fig. 7).

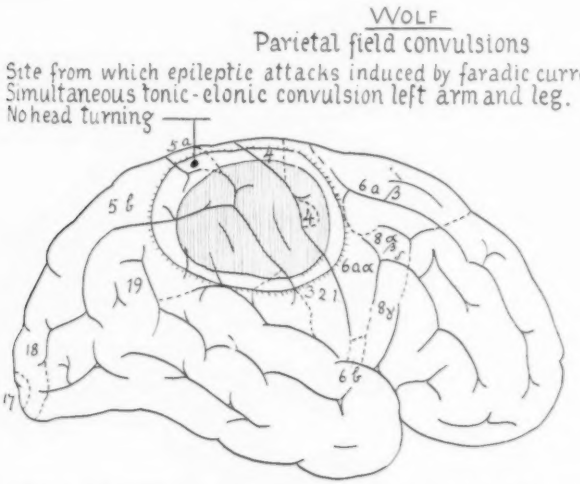

FIG. 8.—*Case* 2. Diagram from operative exposure. Jagged line indicates extent of cicatricectomy. Spontaneous attacks were preceded by sensory aura in the left leg or simultaneously in left leg and arm followed by head, eyes and body turning to left with tonic-clonic spasm of left arm and leg and rapid spread to extremities of right side.

Craniotomy in April, 1928.—Underneath the defect the dura was adherent to a cerebral scar; in the anterior part of the scar was a cyst. Posterior to this the tissue was hard and rather grey. Electrical stimulation near this indurated area produced a convulsion characterized by almost simultaneous movements of the left arm and leg, without head turning (fig. 8). A radical incision was carried out as indicated. Since the operation up till the present time (April, 1930) he had no more attacks.

The gross specimen showed dense adhesion of firm tissue to the under surface of the dura, and a large vessel near the point at which the stimulus had produced the convulsion.

Microscopical examination showed that the indurated areas contained

strands of connective tissue and blood-vessels passing downward from the dura. Some of the vessels were surrounded by sclerotic rings of fibrous neuroglia. There was progressive destruction of tissue taking place as evidenced by pigment-laden cells around certain of the vessels, and also by isolated areas in which the microglia had undergone a partial alteration in the direction of compound granular corpuscles.

Case 3.—The patient received a gunshot wound in the right occipital region in 1914. A preliminary operation was done at once but the patient suffered from headaches and vertigo for some time, and he has had a left-sided homonomous hemianopsia since that time.

In 1916 epileptic seizures appeared, each attack being ushered in by a

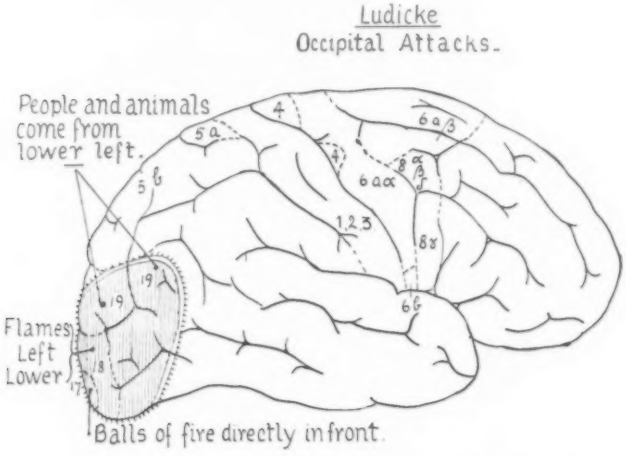

FIG. 9.—*Case* 3. Area of lesion seen at operation indicated by shading. Dots show points stimulated by electrode. The symptoms experienced by the patient following each stimulus are indicated on the chart.

sensation of light in the left visual field. This was followed by turning of the eyes to the left, followed by turning of the head to the left and convulsive movements of the limbs of the left side, ending eventually in loss of consciousness.

Encephalography was carried out in 1927. This showed the posterior horn of the right lateral ventricle to be somewhat enlarged, to be displaced to the right and evidently to communicate with a cyst in the vicinity of the cranial defect. Both ventricles, even as far forward as the thalamus, were displaced somewhat to the right.

Occipital craniotomy was carried out in January, 1927. The dura was found to be densely attached to the cerebral cicatrix in the occipital lobe. Electrical stimulation of field 19 (see fig. 9) caused people and animals to be

seen in the left field, and flames or balls of fire were produced by stimulation in field 18 nearer the occipital pole.

The whole area was radically excised; the epileptic seizures became less

FIG. 10.—*Case* 4. Encephalogram (brow up) eleven years after shell wound of right frontal lobe. Lateral ventricles pulled to right and upwards towards the scar. The radiogram has been reversed in reproduction.

FIG. 11.—*Case* 4. Encephalogram (right side of head up) showing cranial defect with enlargement of anterior horn of the right lateral ventricle, and deflection upward of parts of both anterior horns.

frequent and less intense but did not disappear entirely. One complicating factor in this case is the fact that the patient acquired syphilis in the year following the onset of his epilepsy.

In the excised cerebral cicatrix were two pieces of bone and a remarkable amount of connective tissue.

Case 4.—In 1914 the patient received a shell wound in the right frontal lobe. There was apparently no loss of consciousness and an immediate operation was performed. One year later the cranial defect was closed. At the end of four years, in 1918, the patient began to suffer from epileptic attacks which were of the type of those arising in the frontal adversive field. The eyes and head turned to the left without any sensory aura. This was followed by movements of both the left extremities.

Encephalography which was carried out in 1925 showed that the right lateral ventricle was somewhat larger than the left and was displaced upward

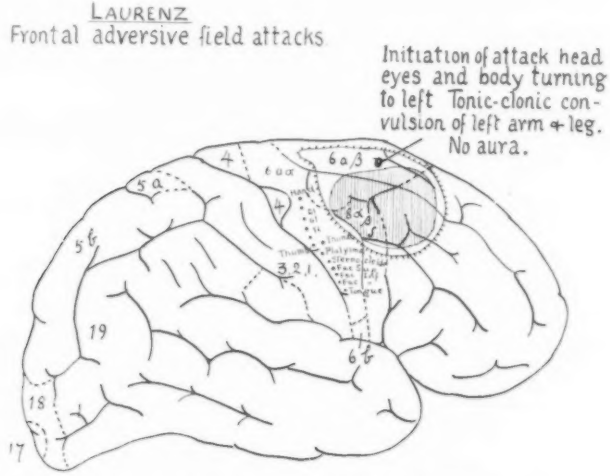

FIG. 12.—*Case* 4. Lesion as seen at operation. Motor response to galvanic stimulation indicated by small dots. Point at which convulsion was induced by faradic stimulation indicated by large dot.

and laterally. The third ventricle was displaced to the right and the left lateral ventricle seemed to be tipped, as though there had been a pull upward toward the cranial defect in the right upper frontal region (fig. 10). In the lateral view (fig. 11) the enlargement and upward deviation of the right lateral ventricle is easily seen.

Frontal craniotomy was carried out in 1925. A dense adhesion between the dura and cerebral cicatrix was found. Electrical stimulation of the brain at the border of the cicatrix (fig. 12) initiated an attack which was quite typical of those from which the patient suffered ordinarily.

The operative recovery was satisfactory, and during the three years which followed the operation there were no further convulsions.

The excised tissue contained yellowish grey gelatinoid material as is usual

in these cicatrices. Two bits of shell were found embedded in the tissue. Histologically there was a large amount of gliosis and remarkable parallelism of the glia fibres. There was a very well-developed vascular plexus entering the scar (fig. 13).

Case 5.—In 1916 the patient received a gunshot wound in the parietal region near the vertex, a little more on the left than the right. An immediate operation was carried out. There followed a bilateral spastic paresis, more marked on the left side and associated with sensory disturbances of both legs.

In 1923 he began to have epileptic seizures typical of those arising in the right parietal adversive field. The head and eyes turned towards the left and

FIG. 13.—*Case* 4. Plexus of vessels and collagen fibres entering brain from overlying cicatrix. Silver carbonate connective tissue stain.

then suddenly simultaneous movements appeared in the left arm and leg, followed quickly by the right leg.

Encephalography showed some enlargement of the lateral ventricles and an upward deviation of the right side.

Parietal craniotomy was carried out and a large meningocerebral cicatrix found. The operation was carried out in two stages. At the first operation during the dissection of the scalp free from the dura, several typical convulsions were produced apparently as a result of the manipulation of the scar.

At the second stage of the operation electrical stimulation of the brain in the vicinity of the scar produced a typical attack. A wide excision was carried out as indicated (fig. 14). The excised tissue showed the usual type of con-

tracting cicatrix. During the four years which have elapsed since operation the patient has been completely free from any attacks.

Case 6.—The patient gave an obvious history of birth injury followed by

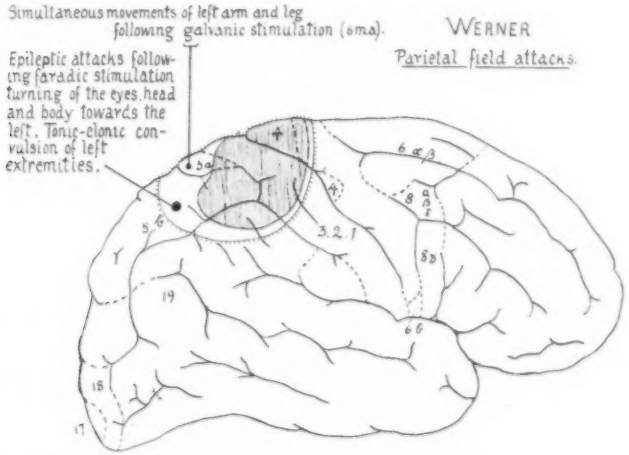
Simultaneous movements of left arm and leg following galvanic stimulation (6 ma).
WERNER
Parietal field attacks.
Epileptic attacks following faradic stimulation turning of the eyes, head and body towards the left. Tonic-clonic convulsion of left extremities.

FIG. 14.—*Case* 5. Location of lesion and extent of removal.

FIG. 15.—*Case* 6. Encephalogram (brow up) nineteen years after birth injury. Cyst of left hemisphere. The radiogram has been reversed in reproduction.

right-sided hemiplegia which gradually improved during boyhood. There was normal mental development up to the age of 12, after which time he was somewhat retarded. At 14 he began to suffer from epileptic seizures which began

with movements of the right arm and leg, and simultaneous turning of the head and eyes to the right, followed by a generalized convulsion.

At the age of 19 encephalography showed a large cyst in the left cerebral hemisphere and some enlargement of the ventricle on that side (fig. 15).

Craniotomy exposed two large cysts close together. Electrical stimulation

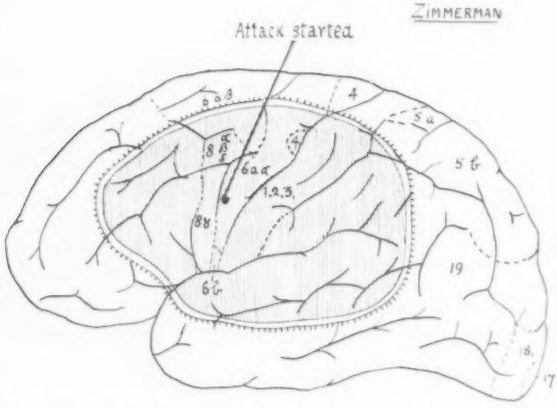

FIG. 16.—*Case* 6. Extent of lesion as seen at operation shown by shading. Extent of removal indicated by jagged line. Faradic stimulation at large dot between cysts inaugurated an attack.

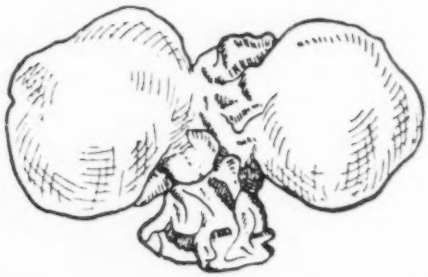

FIG. 17.—*Case* 6. Sketch of excised tissue after formalin fixation showing the two cysts.

between the cysts (fig. 16) produced a typical convulsion. Both cysts were excised together with an indurated intravening area of cicatrix (fig. 17). The tissue of this scar presented a curious appearance (fig. 18), being made up for the most part of strands. These strands were composed of neuroglia cells with long parallel fibres, each strand being surrounded by a coating of collagen. The arrangement resembled that seen in peripheral nerves.

The patient had been entirely free from epileptic seizures when last heard from three years after the operation.

FIG. 18.—*Case* 6. Cross section of the two cysts and the intervening cicatrix. Note the fibrous strands.

FIG. 19.—*Case* 7. Encephalogram (brow up) nineteen years after birth injury, showing enormous enlargement of right lateral ventricle and some pulling of the third ventricle across toward the right side.

FIG. 20.—*Case* 7. Encephalogram (right side up) showing cranial defect from previous exploration and enlargement of anterior horn of right lateral ventricle.

Case 7.—This is a second case of cerebral birth injury followed this time by left-sided hemiparesis. At the age of 9 he began to suffer from epileptic attacks typical of those arising in the right frontal adversive field. The head and eyes turned to the opposite side followed by convulsive movements of the opposite extremities. The following year an opening was made in the bone over the right frontal area without entering the dura. This was without effect on the epilepsy.

Encephalography in 1928, at the age of 19, showed enormous dilatation of the right lateral ventricle with some displacement of the third ventricle across

FIG. 21.—*Case* 7. Low-power photomicrograph of strands of cicatrix in cross section. Collagen makes a dark line about each. The dots within are neuroglial nuclei.

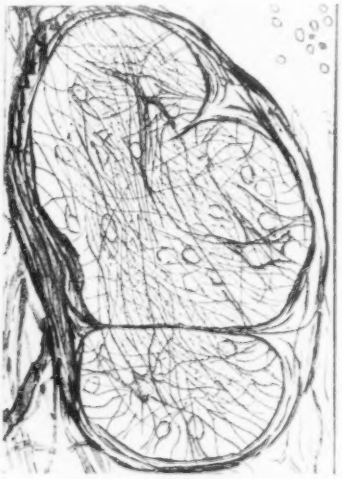

FIG. 22.—*Case* 7. High-power sketch of one of the strands seen in fig. 21. Some of the astrocytes can be seen to apply large processes to the collagen lining. The fine fibres are all neuroglial and arise from these cells. The dots indicate numerous such fibres cut across. Silver carbonate astrocyte stain, Hortega.

to the right side (fig. 19). The enlargement was chiefly in the anterior end of the ventricle (fig. 20).

Craniotomy the same year demonstrated a dense adhesion between the dura and the brain scar. Dissection of the scar produced a typical attack. The lesion was excised, thus removing the lateral wall of the ventricle.

This excised tissue like that examined in the last case contained strands of glial tissue in which no nerve-fibres were to be found. The neuroglia cells were fibrous, containing many long, slender neuroglia fibres. These strands were coated with collagen and the neuroglia cells within were attached by numerous

footplates to the under surface of the collagen covering. This is tissue capable of withstanding a considerable pull. These strands (fig. 21 and 22) were unlike any other type of pathological material. The tissue can hardly be called cerebral tissue in the strictest sense, inasmuch as there are no nerve-cells and no nerve-fibres in it, and although the tubes are composed entirely of neuroglia they have been coated over with collagen, just as nerve-fibres in other portions of the nervous system are invariably covered.

Operative Technique.

The general preparation and pre-operative study are indicated in the outline of the above cases. Operation must be carried out under local anæsthesia. After electrical exploration radical excision is practised.

The removal should go deep enough to carry away all of the injured tissue and is best carried out by passing sutures deep down into the brain and tying them one by one so that the suture itself cuts through the cerebral tissue and procures hæmostasis of the blood-vessels in the pia arachnoid. The defect in the dura should be closed by the insertion of a piece of fascia. Operations that provide for the closure of the bone defect without removal of the scar can hope to accomplish nothing. Likewise dissection of the dura free from the scar and the interposition of an implant should only make matters worse, for no matter what the implant may be, it stimulates the formation of increased amounts of connective tissue. Only radical excision can remove the scar and relieve the abnormal physical strain. Anything less will serve only to increase the cicatrizing process.

There are minor differences in operative procedure as carried out by the two authors. In the neuro-surgical clinic at Breslau the bone is removed permanently over the area of operative exploration, while in Montreal the original procedure has been modified to the extent of replacing the bone flap The important feature is that the removal is radical, clean, and directed toward the lesion responsible for the attacks.

Discussion.

There were five additional cases included in the original report which will not be cited here: the ones already taken up being sufficient to serve as examples. These cases were selected because they illustrated certain aspects of the problem of traumatic epilepsy. Numerous similar cases could be added from the experience of each of the authors.

In each of the twelve cases studied there was displacement of one or all of the ventricles towards the side of the lesion. In most of the cases there was some degree of enlargement of the ventricle on the side of the lesion. In the two cases of birth injury the enlargement was enormous. That the local atrophy is not the cause of the deflection of the ventricles towards the site of the lesion is shown by the fact that the wandering of the ventricles is not greater in those cases where the atrophy is great. Likewise cases of cerebral thrombosis do not show

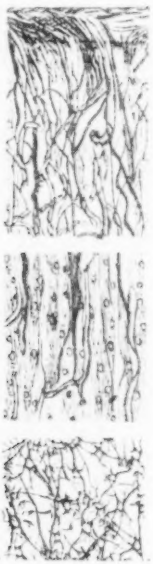

FIG. 23.—Schematic drawing of three levels in any meningo-cerebral cicatrix of long standing. Uppermost panel shows blood-vessels and bundles of collagen penetrating the brain beneath the meninges. Middle panel from area farther below surface shows intimate relationship of collagen strands and fine-fibred neuroglia, all parallel and pointing in the direction of the cicatricial pull. Lowermost panel from an area deeper in the brain shows the structure of astrocytes and blood-vessels which make up the vaso-astral framework and transmit the cicatricial pull.

this ventricular displacement and many cases with little or no enlargement of the lateral ventricle may show quite marked ventricular displacement. Instead of wandering of the ventricles the process is really brain-pull or ventricle-pull.

Its seems evident that cicatricial contraction is responsible for the displacement of the ventricles in these human cases just as has already

been demonstrated experimentally. Histological examination of this human material provides eloquent evidence of a long-continued cicatricial traction. There was always fibrous tissue, especially near the surface, and adhesion to the meninges (fig. 23). Thus connective tissue and an astonishingly rich plexus of vessels were invariably present in the scars, intermingled with fibrous astrocytes whose fibres were in general arranged in parallel and extended in the direction of the obvious traction, that is upward toward the cicatrix (fig. 23, middle panel). Deeper down in the brain (fig. 23, lowest panel) the astrocytes and blood-vessels still continue to form the only framework capable of withstanding tension, the vaso-astral framework [1].

In the areas of gliosis nerve-fibres are rare and nerve-cells still rarer. Occasionally localized areas are found in which there are groups of phagocytes containing pigment. Such patches are evidently in the vicinity of blood-vessels which have been shut off and they seem to indicate that the process of cerebral destruction is a long-continued one as they were present twelve and fourteen years after the initial wound. These areas recall the focal perivascular destruction described by Spielmeyer [3] in epilepsy of a different type.

The most extreme degree of progressive scarring is seen in the two cases of birth injury, both of which were of nineteen years standing. In these cases the process of connective tissue invasion and fibrillation of neuroglia had gone to such a point that there were tubes and sheets of neuroglia surrounded by connective tissue sheaths. Even where local atrophy and destruction had given rise to the presence of cysts there was always to be found also an area of grey, gelatinous fibrous tissue, the centre or the focus of concentric traction.

One further convincing evidence of the physical pull which these scars exert is the fact that in one of these cases reported here and in a number of others which we have observed, there was a definite pulling in of the scar which was noted as soon as the bone was removed and the edges of the adherent dura incised. The centre of the scar thus drew itself together, sucking the attached dura inward as much as 1½ cm. as soon as it was freed from the attachment to the overlying skull.

Relationship of Cicatricial Contraction to Traumatic Epilepsy.

An outstanding feature of all of these scars is the rich plexus of newly-formed vessels in and about the cicatrix. This vascular plexus

anastomoses very freely with the large vessels which enter the scar from without. It also anastomoses freely with intracerebral vessels inasmuch as the surface of the dura continues to bleed after it is completely exposed and cut free from surrounding dura. We have already shown [1] also that experimentally this vascular plexus can be injected either by way of the external carotid artery or through the internal carotid.

During the years which follow the infliction of a brain wound there is evidently a progressive increase in the vascular elements of the scar and a progressive decrease in the nervous elements. Associated with these two changes is the steady cicatricial contraction which obviously takes place for an indefinite period.

Now if the stimulus for an epileptic seizure arises in the nervous elements one might expect the attack to begin shortly after the infliction of the wound when these elements are plentiful and undergoing progressive destruction. If, on the other hand, the vascular elements are responsible for the initiation of the process, epilepsy might well be expected to make its appearance later when the vascular plexus reaches its fullest development and nerve-fibres have largely vanished.

In the twelve cases reported, the time between the wound and the onset of convulsion varies between five months and fourteen years, the average period of immunity before the onset of attacks being five years and six months. The acute effect of the injury must have disappeared long before the end of the fifth year. There remains, however, one process progressive through all these years, and that is the cicatricial contraction which gradually draws one or both hemispheres toward the lesion.

We have shown that at operation the focal epileptic attacks may often be produced in two ways: either by electrical stimulation of the brain in the neighbourhood of the wound, or by gently pulling upon the adherent dura. This latter fact may be of considerable significance, for if increase of a pre-existing strain produces an attack it may well be that the pre-existing strain itself is an important factor in the ætiology of spontaneous convulsions.

As pointed out above, the blood-vessels form in one sense the woof of the contracting network. Traction, therefore, upon the vessels must be inevitable. The hypothesis at once suggests itself that a vaso-motor reflex secondary to this traction is responsible for the initiation of the convulsive seizures.

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THE ANTERIOR CEREBRAL ARTERY, AND ITS SYNDROMES.

BY MACDONALD CRITCHLEY.

Comparative Anatomy.

Throughout the animal series the anterior cerebral artery has varied considerably in uniformity and has tended to become more complex with the mammalian ascent. Closely associated with the type of anterior cerebral artery has been the conformation of the forward portion of the circle of Willis.

The most primitive pattern is encountered amongst the fishes, amphibians, reptiles and birds. Here there is no real circle of Willis, as no anterior communicating artery occurs. The two anterior cerebral arteries are represented by a pair of small vessels lying parallel on the mesial aspects of the olfactory lobe. Hofmann [35] has shown that in the fish the anterior cerebral artery in its forward course sends small twigs to the under-aspect of the frontal lobe and terminates in the mesial aspect of the hemisphere in a series of small vessels. Although no anterior communicating artery is the rule, Schöbl [53] has described a ramus communicans in the salamander. In snakes, tortoises and crocodiles the anterior cerebral artery of each side may unite to form a common azygos artery from which arise the main vessels to the olfactory bulbs. Rathke proposed the name arteria ethmoidalis communis for this vessel.

Amongst birds the anterior cerebral artery is represented by a very small paired vessel having no communication with its fellow. The true ramus cranialis of the carotid artery is constituted by a large ethmoidal artery which passes forwards through the ethmoidal foramen.

Mammals.

In the giant ant-eater one encounters the primitive avian type, with small paired arteries and no ramus communicans; there is, therefore, no true circle of Willis.

The typical mammalian pattern consists in two short anterior cerebral arteries which converge and unite to form a median azygos

vessel; this may or may not bifurcate later. There is no anterior communicating artery. The chief territory of the anterior cerebral artery and its branches is the forward portion of the rhinencephalon. Shellshear [60] has recently made a minute study of the morphology of the anterior cerebral artery in the spiny ant-eater. Here, the circle of Willis is completed in the usual mammalian fashion. The azygos

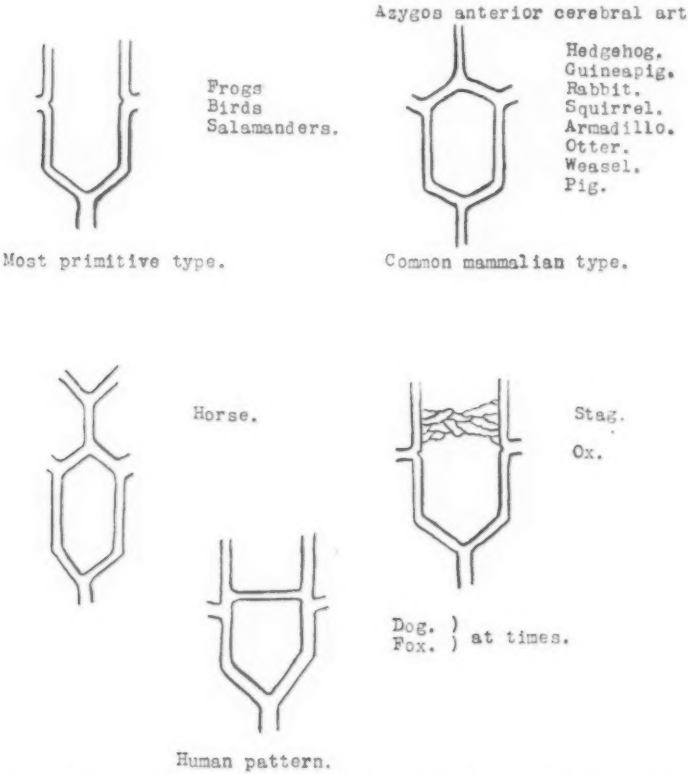

FIG. 1.—Schematic representation of the circle of Willis in animals lower than monkeys.

vessel lies in the median cleft of the brain between the two tubercula olfactoria, and terminates by dividing into a number of branches to the medial surface of the bulb and neopallium, including vessels to the anterior perforated spot, the postero-medial region of the tuberculum olfactorium and the area præcommissuralis; finally a large branch passes outwards to anastomose with a branch of the middle

Sylvian artery. In this way a vascular ring is formed around the tuberculum olfactorium. Shellshear identifies this particular branch with the so-called artery of Heubner in the human brain.

In mammals possessing a corpus callosum, the azygos anterior cerebral artery usually divides after a variable distance into two small "arteries of the corpus callosum" which run side by side over the dorsum of that structure.

The course of distribution of the anterior cerebral artery in the cat may be studied rather more closely, as typifying the usual lower mammalian arrangement. Shortly after the junction of the two arteries immediately below the genu of the corpus callosum, two vessels are given off, one to each hemisphere. The branch to the left cerebrum

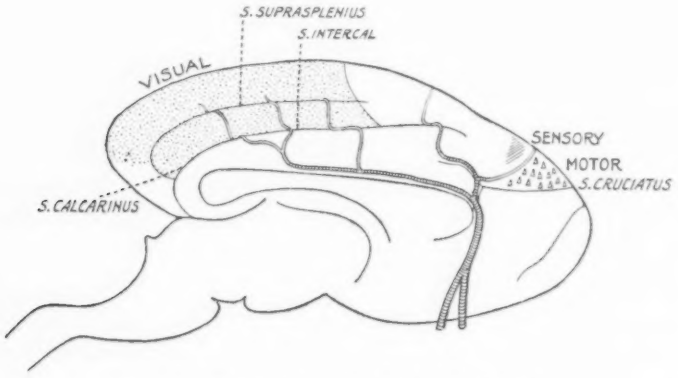

FIG. 2.—The anterior cerebral artery in the cat.

emerges before the one to the right side. The vessel ascends directly upwards in the vertical limb of the sulcus cruciatus, where it divides into anterior and posterior branches (see fig. 2). The anterior division proceeds to the motor and sensory areas, while the posterior branch turns backwards and then upwards over the most anterior portions of the sulcus splenialis to the parietal region. The azygos anterior cerebral artery now divides into two arteriæ corporis callosi which pass backwards in close apposition over the dorsum of the corpus callosum. Midway between the genu and the splenium, a second branch crosses the gyrus fornicatus, bends backwards for a short way in the sulcus splenialis and then, after making a second bend, runs upwards into the foremost extremity of the sulcus suprasplenialis. The terminal portion leaves the corpus callosum just anterior to the splenium and runs

upwards and backwards. Before entering the sulcus splenialis it divides into anterior and posterior branches. These also run upwards parallel to each other and terminate in the mesial aspect of the visual area, in the gyrus suprasplenialis and the gyrus postsplenialis.

In the horse the unpaired portion of the anterior cerebral artery divides in the region of the genu into four terminal branches, two of which are large and two are small. The two smaller vessels, the arteriæ corporis callosi, run parallel over the genu and dorsum of the corpus callosum to terminate in the region of the splenium. The larger vessels

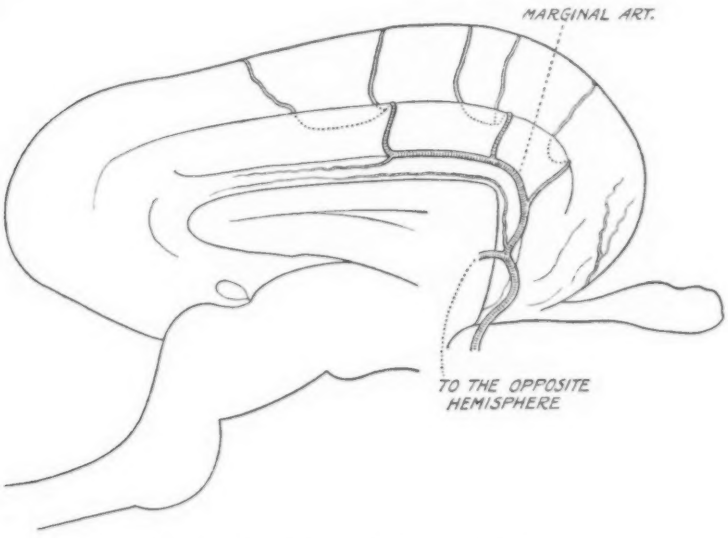

FIG. 3.—The anterior cerebral artery in the horse.

are distributed to the mesial aspects of both hemispheres. Each runs in the sublimbic fissure, whence it gives off two large branches which cross the gyrus fornicatus, and after dipping deeply into the calloso-marginal gyrus, terminates in the superior border of the convex surface. (See fig. 3). The main trunk of the vessel leaves the sublimbic sulcus at about the middle of the corpus collosum and terminates by dividing within the depths of the calloso-marginal fissure into two vessels. A small arteriole arises from the point of angulation and passes backwards in the callosal sulcus. These two larger vessels represent the marginal artery.

A still greater disproportion is seen in the brain of the calf, between the size of the arteries of the corpus callosum and the marginal vessels. In the particular specimen examined there was an anterior communicating artery connecting the two anterior cerebral arteries. Immediately in front of this was a second and finer anastomotic vessel. The anterior cerebral artery then divided into a fine artery of the corpus callosum, and a large marginal artery. The latter turned forwards in the mesial aspect of the frontal lobe and then bent back so as to enter the callosomarginal fissure. Immediately before entering this sulcus a large anterior branch is given off to end in the sulcus coronarius (see fig. 4).

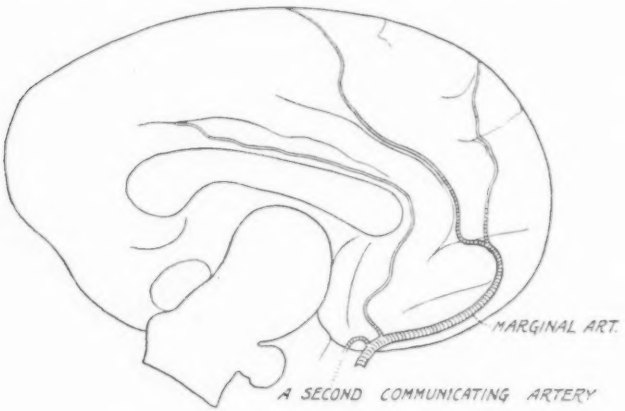

FIG. 4.—The anterior cerebral artery in the calf.

Amongst the lower monkeys (baboons, macacques, long-tailed monkeys) there is a great variability in the pattern of the anterior aspect of the circle of Willis and numerous transition forms are encountered between the mammalian and human types. Thus, in a series of thirty-two lower monkeys examined by Rothmann [52], a primitive mammalian type of anterior cerebral artery was found in twenty (i.e., an azygos vessel, and no anterior communicating artery); in five cases a small vascular loop occurred at the point of junction of the paired and unpaired vessels, and in one instance there was a double loop. In two cases the paired portion of the anterior cerebral artery was well developed on one side only; in another case the azygos vessel arose from the internal carotid artery. In only three instances was there any suggestion of a human pattern of an atypical type.

A more detailed study of the branches of the anterior cerebral artery in the baboon shows an obvious similarity with the human pattern (see fig. 5). Thus, the first branch passes horizontally forwards to supply the gyrus rectus. Immediately below the genu a large branch passes forwards and upwards to enter the deep calloso-marginal sulcus, whence it emerges to pass to the cortex as high as the supero-mesial border. It supplies the internal aspect of the frontal lobe as far posteriorly as the midfrontal sulcus. The third branch is given off just behind the genu; it runs parallel with the previous branch, enters the depths of calloso-marginal sulcus, and emerging thence passes to the uppermost part of the lateral surface. It supplies the area between the midfrontal sulcus and the fissure of Rolando. The fourth branch arises

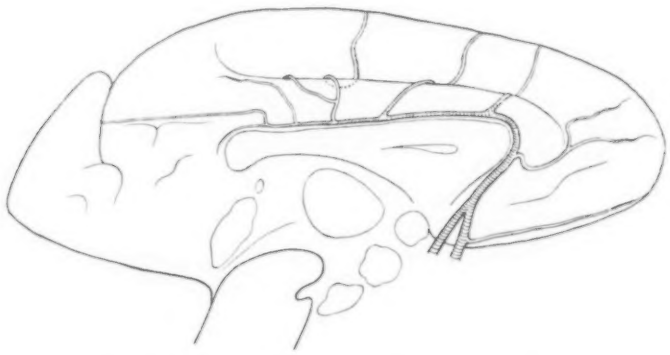

FIG. 5.—The anterior cerebral artery in the baboon.

about the centre of the corpus callosum. It passes upward and slightly forwards, dipping deeply into the calloso-marginal fissure, and ends in the area surrounding the mesial prolongation of the fissure of Rolando. A fifth branch is given off at the junction of the posterior third with the anterior two-thirds of the corpus callosum; just before entering the calloso-marginal sulcus it gives off a posterior branch which communicates with the sixth branch. The main stem enters the calloso-marginal sulcus and emerges at a little distance anteriorly, whence it passes directly upwards to supply the post-central cortex. The sixth branch does not enter the calloso-marginal sulcus but turns back to run below and parallel with it as far as the precuneus. The terminal portion of the anterior cerebral artery turns abruptly up at the level of the splenium and then almost immediately changes direction so as to run

backwards and slightly downwards to end in the parieto-occipital fissure. A tiny twig continues the direction of the main artery over the splenium and anastomoses with a twig from the posterior cerebral. These details of the branches of the anterior cerebral artery in the baboon agree in the main with the description given by Lesem [37]).

The arterial patterns in the higher apes show a greater tendency towards a human type than in the other mammals. In the chimpanzee, Rothmann found a typical human arrangement in one out of seven; in one there were two anterior cerebral arteries but no anterior communicating artery. In four the conformation was of the primitive mammalian type, whilst in one there were three anterior cerebral arteries with an arterial loop at the point of trifurcation. Grünbaum and Sherrington [31] discovered a human pattern in five out of six chimpanzees, while in one only was there an azygos anterior cerebral artery. In one chimpanzee brain, studied in great detail by Shellshear [61], the vessel was of the mammalian type. His work reveals a striking similarity in the distribution of the deep and superficial branches between the chimpanzee and the human brains. Professor Elliot Smith has very kindly permitted me to read the manuscript of Shellshear's paper.

No anterior communicating artery was found in either of the two orang brains examined by Bolk [16]. A characteristic human conformation existed in the brain of one orang examined by Grünbaum and Sherrington. In Rothmann's four cases no typical specimen was found, either of the human or of the mammalian type. In two, there was an asymmetry between the two sides; in one there were three anterior cerebral arteries, and in another there were numerous anastomoses between the two anterior cerebral arteries. Shellshear [58] has studied minutely the morphology of the cerebral blood-vessels in an orang brain. In this particular specimen, the circle of Willis was completed in the normal human fashion. The branches from the first part of the artery, that is from its origin to the anterior communicating vessel, supplied the anterior commissure, the corpus striatum lying in front of the anterior commissure, the olfactory tract, the mesial and lateral olfactory striæ, the tuberculum olfactorium, the corpus paraterminale and the proximal part of the gyrus cinguli. One branch from this region was conspicuous on account of its size; it probably supplied the caudate nucleus. Branches from the second part were purely cortical; the first, corresponding with the medial orbital branch in man, supplied the area præfrontalis; the second branch was called the anterior medial frontal

and was distributed to the area frontopolaris. The third branch—which is often associated with the second—supplied the anterior frontal area of Elliot Smith. The fourth and fifth branches were termed the intermediate and posterior medial frontal respectively, and irrigated the superior frontal and paracentral gyri. Other twigs supplied the posterior part of the gyrus cinguli and the area parasplenialis. The anterior cerebral artery terminated in the parieto-occipital fissure.

The brains of two gorillas in Rothmann's series both showed a typical human formation.

Out of four brains of gibbons, Rothmann found a mammalian pattern three times. In a fourth there were two anterior cerebral arteries, but the anterior communicating artery was replaced by a large vascular knot.

Anatomy of the Anterior Cerebral Artery in Man.

In the human anterior cerebral artery a still greater degree of complexity is reached, although the main conformation resembles closely that seen in the higher apes.

Arising as one of the two terminal branches of the internal carotid, the anterior cerebral artery first passes forwards and inwards across the anterior aspect of the anterior perforated spot. It crosses between the tuberculum olfactorium and the optic nerve and so comes to lie at the margin bounding the mesial and orbital surfaces of the hemisphere. At this point the two arteries are connected by the short anterior communicating artery. The anterior cerebral artery then abruptly changes its direction and turns upwards and slightly forwards so as to reach the genu of the corpus callosum. It now follows the epicallosal sulcus and lies along the body of the corpus callosum as far as the junction of the posterior fifth with the anterior four-fifths. In this way it courses forwards, then upwards, backwards and slightly downwards, and finally pursues an almost horizontal course. The artery terminates by leaving the epicallosal sulcus, and passing upwards and backwards obliquely across the parietal lobe, ends either in the quadrate lobe or in the parieto-occipital fissure. Although the two anterior cerebral arteries lie in close apposition throughout their courses there is, in the majority of cases, no intercommunication after the anterior communicating artery.

The branches of the anterior cerebral artery may be classified as follows :—

(1) Basal (arising from the artery as it crosses the anterior perforated spot).

(2) The anterior communicating artery.

(3) The branches from the convexity.

(4) The branches from the concave aspect of the arterial course.

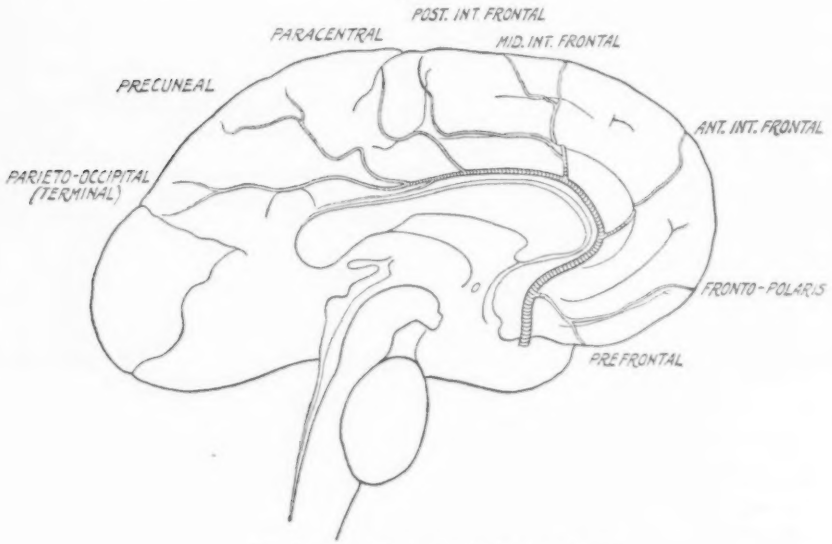

FIG. 6.—The anterior cerebral artery in man.

(*a*) *The basal branches.*—Although most French writers have minimized the importance of these vessels, the work of Heubner [33], which has been abundantly confirmed by others, illustrates their constancy and importance. According to the French school, the fine basal branches pierce the anterior perforated spot to supply the head of the caudate nucleus, but the basal ganglia and converging motor pathways are said to receive no supply from the anterior cerebral artery.

The fine basal branches, usually three or four in number, pass immediately upwards, parallel with one or two similar vessels from the region of the bifurcation of the internal carotid, and enter the anterior perforated spot through the most anterior row of apertures. They are distributed to the head of the caudate nucleus, and in particular to its anterior-inferior portion. At times the whole of its head may be so supplied.

One of the basal perforating branches of the anterior cerebral artery is important, however, on account of its size, its course and its distribution. This vessel—the recurrent artery, or the artery of Heubner [1]—emerges from the superior surface of the anterior cerebral artery, sometimes in the most mesial portion of the anterior perforated spot, sometimes at the lateral end. It may occasionally arise from the internal carotid at its bifurcation, or even from the middle cerebral. I have also seen it spring from the anterior cerebral artery after the emergence of the anterior communicating artery. According to Aitken [2], however, it arises in 80 per cent. of cases from the basal portion of the anterior cerebral artery. This vessel passes first downwards, and then almost immediately turns outwards and backwards towards the lateral extremity of the anterior perforated spot, which it pierces either as a single or as a double vessel. Before entering the substance of the brain it may occasionally send off a fine twig to the tuberculum olfactorium and the proximal portion of the olfactory peduncle. Within the brain it passes upwards and outwards, branching into three main stems, and ends in the anterior part of the caudate, the anterior third of the putamen, the tip of the outer segment of the globus pallidus, and the anterior limb of the internal capsule (see figs. 7, 8, 9, 10 and 11).

Another of the basal arteries, according to Aitken, constantly enters the anterior perforated spot through a large, centrally placed aperture and is distributed to the globus pallidus only.

(*b*) *The anterior communicating artery* is a short vessel (about 4 mm. in length) connecting the anterior cerebral arteries of both sides. It passes horizontally backwards, lying immediately above the optic nerves and in front of the chiasma. In man there are no branches, although when present in dogs it may send fine twigs to the stalk and the anterior lobe of the pituitary. (Dandy and Goetsch [20].)

(*c*) *The branches from the convexity* are the largest and most important.

These are variable in their mode of origin and two or three may issue from a common stem. There is a striking lack of uniformity in nomenclature, particularly on account of the survival of Duret's original terminology. Duret [21] described one chief branch which he named

[1] The artery is named after Heubner, who, in 1874, described a vessel emerging from the anterior cerebral artery which supplied the forelimb of the inner capsule, as well as the most internal and the middle lobe of the lenticular nucleus. Despite this work and the later researches of Beevor, Aitken and others, the French have continued to ignore its existence and to associate themselves with the original findings of Duret.

"the internal and inferior frontal artery," and he stated that the anterior cerebral artery terminated by dividing into three vessels, namely, an "internal and anterior frontal artery," the "internal and medial frontal artery," and the "internal and posterior frontal artery."

In this paper the terminology employed by Shellshear is largely adopted, with one or two modifications. The branches may be described as follows:—

(1) *The prefrontal branch* (syn., the orbital artery of Foix; the internal orbital artery).—This vessel arises almost immediately after the emergence of the anterior communicating artery. It bends downwards

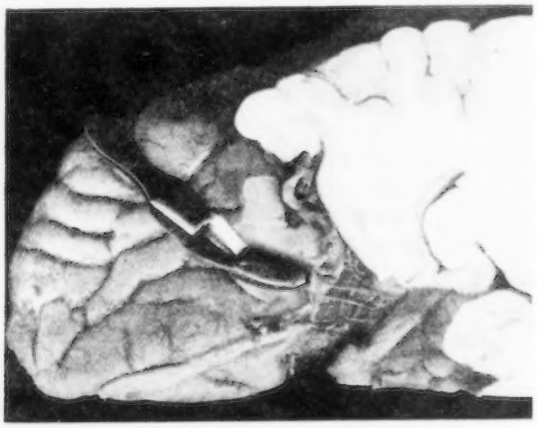

FIG. 7.—The anterior perforated spot of the left hemisphere of the human brain. The internal carotid artery has been turned forwards to display the first parts of the anterior and middle cerebral arteries. Three perforating branches of the anterior cerebral artery are seen passing backwards to enter the perforated spot. Heubner's artery runs transversely outwards above the perforating branches, and after dividing into two, enters the brain at the lateral extremity of the perforated spot. See also fig. 8.

to the orbital surface of the frontal lobe and crosses obliquely the gyrus rectus. Here it frequently divides into two branches which pass dorsal to the olfactory peduncle, at the junctions of the anterior one-third and posterior two-thirds, and the anterior two-thirds and posterior one-third respectively. The prefrontal artery supplies the medial aspect of the frontal lobe below the sulcus subrostralis, the gyrus rectus, the olfactory peduncle and lobe, and the internal orbital convolution; it reaches as far forwards as the tip of the pole. Its territory corresponds closely with the *area præfrontalis* of Elliot Smith, or Brodmann's area, No. 11.

Not infrequently the prefrontal artery arises as a branch of the frontopolaris artery.

(2) The *frontopolaris branch* (syn., the prefrontal artery of Foix; the internal anterior-frontal artery of Duret; the internal frontal artery of Lesem).—This vessel arises midway between the inferior border of the mesial surface of the hemisphere and the genu of the corpus callosum. The artery passes slightly downwards at first and then horizontally, lying in the sulcus rostralis. It terminates in the tip of the frontal pole where it supplies the most anterior portion of the superior frontal gyrus. This vessel possesses the most direct course of all the branches of the anterior cerebral artery. The subcortical territory of this vessel, as revealed by a horizontal section through the frontal

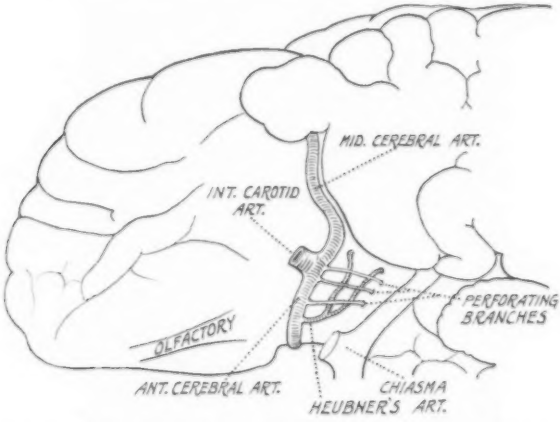

FIG. 8.—The vessels to the anterior perforated spot depicted semi-diagrammatically.

pole, consists in a rectangular area measuring $2\frac{1}{2}$ by 3 cm., bounded anteriorly by the cortex of the frontal pole, intermediately by the cortex of the mesial surface, and posteriorly by the tip of the anterior horn of the lateral ventricle.

In man its territory corresponds with the *area frontopolaris* of Elliot Smith or the medial aspect of Brodmann's field No. 10.

(3) The *anterior internal frontal branch*. This branch emerges at the level of the genu. It passes forwards and upwards across the gyrus cingulus, to enter the depths of the calloso-marginal gyrus and terminate in the convex surface of the brain in the middle portion of the superior frontal convolution. It supplies the mesial portion of Brodmann's field No. 9 or the *area frontalis granularis*.

(4) The *middle internal frontal* arises at the level of the body of the corpus callosum and passes upwards to enter the depths of the calloso-marginal gyrus. After making a loop it emerges and passes up and slightly backwards. It frequently bifurcates before entering the convexity of the cerebrum, where it ends in the upper or posterior end of the superior frontal gyrus. The territory corresponds with the mesial portion of Brodmann's field No. 8, or the *area frontalis intermedia*.

FIG. 9.

(5) The *posterior internal frontal branch* often emerges from a point just behind the former artery and runs upwards to the calloso-marginal sulcus. It then passes backwards in this fissure for a distance of 1 to 1·5 cm., when it again turns upwards, and ends in the uppermost limit of the precentral fissure. The area of its supply is roughly the medial aspect of Brodmann's field No. 6, or the *area frontalis agranularis*.

The three internal frontal arteries frequently do not leave the anterior

cerebral artery by three stems, but may be combined; thus the anterior and middle may emerge from a common stem, or the posterior internal frontal may arise as a branch of the middle internal frontal. A particularly common mode of origin is for a vessel to leave the main trunk of the anterior cerebral artery at the level of the genu and to run forwards as far as the calloso-marginal fissure. Here, the vessel makes a right-angle bend and passes backwards in the depths of this fissure for a distance of 3 to 5 cm. It then makes a second bend at a right-angle

FIG. 10.

and crosses the marginal gyrus to reach the superior frontal convolution. In its course it gives off one, two or three branches; this vessel—known as the *calloso-marginal branch*—represents the fusion of two or more internal frontal arteries, including at times the paracentral artery. The calloso-marginal branch is probably the homologue of the marginal artery of the horse and cow; it is interesting to note how the disproportion which exists in these animals between the arteriæ corporis callosi and the marginal artery, is not encountered in man, owing to the relative greater importance of the corpus callosum.

At times all three frontal branches may arise as trifurcations of a single artery.

(6) The *paracentral artery* arises from a point about the middle of the corpus callosum and passes backwards and upwards to run for a short way in the calloso-marginal gyrus. It then changes its course again to run vertically upwards, ending in the substance of the para-

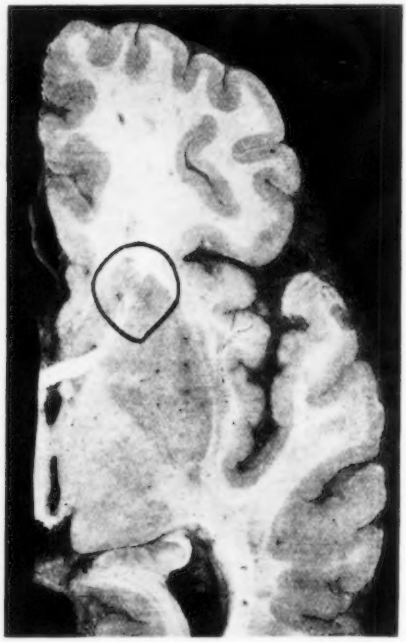

FIG. 11.

FIGS. 9, 10 and 11.—Dissections of the base of the brain to demonstrate the course and distribution of Heubner's artery. The vessel enters the brain in two divisions which later become three; they are distributed to the head of the caudate nucleus, the most anterior portion of the internal capsule and the head of the putamen.

central lobule. In rare cases, this branch is actually the terminal portion of the anterior cerebral artery.

(7) Occasionally, one, or even two, slender branches leave the anterior cerebral artery immediately behind the paracentral artery, and pass directly upwards to terminate in the post-central cortex and superior parietal convolution. These vessels may be termed the *superior parietal arteries*.

(8) The *precuneal branch* in many cases constitutes the terminal portion of the main trunk of the anterior cerebral artery. It leaves the corpus callosum at the point of junction of the posterior one-fifth and anterior four-fifths, and courses obliquely backwards and upwards across the parietal lobe to end in the quadrate lobule. Beevor [11] [12] found this mode of termination of the anterior cerebral artery in twenty-one of his series of forty-eight cases. Its territory corresponds with the mesial portion of Brodmann's field No. 7, or the *area parietalis superior*.

(9) In many instances (nineteen out of forty-eight: Beevor) the anterior cerebral artery ends as the *parietal-occipital branch*. In this case it leaves the dorsum of the corpus callosum immediately posterior to the precuneal artery and runs horizontally backwards to end in the parieto-occipital fissure.

In a single brain, the anterior cerebral artery of one side may end as a precuneal artery, and in the opposite hemisphere as a parieto-occipital artery.

(*d*) *Branches from the concavity.*—These include numerous short twigs to the genu and body of the corpus callosum. Some of them penetrate and supply the septum pellucidum, part of the anterior commissure and the anterior pillars of the fornix.

One longer but very delicate vessel (the *posterior pericallosal artery*) emerges from the under surface of the anterior cerebral artery between the points of origin of the paracentral and precuneal arteries. It passes backwards in the fissure of the corpus callosum, lying immediately under the main trunk of the anterior cerebral artery, and when this latter has left the fissure to form the parieto-occipital artery it continues along the body of the corpus callosum to unite, over the splenium, with a branch of the posterior cerebral artery.

The curved and backward sweeping course of the anterior cerebral artery and its branches is striking. French writers describe two concentric vascular semicircles: an inner one formed by the main trunk of the anterior cerebral artery and its continuation—the posterior pericallosal; and an outer, interrupted one, formed by the various branches in their passage along the calloso-marginal sulcus. The former is termed the "démi-cercle péricalleux" and the latter the "démi-cercle calloso-marginal."

The depth to which the branches penetrate the calloso-marginal sulcus has already been mentioned, although this feature is not so striking as in the brain of certain of the apes, e.g., the baboon.

Skiagraphy of the brain after the injection of a radio-opaque fluid as employed by Temple Fay [25] demonstrates this looping very clearly. Fay remarked that his negatives illustrate a striking difference in the territory of the anterior cerebral artery and of the other vessels; in the former, there were numerous swing-like loops drooping downward from the cortex into the depths of the convolutions towards the anterior horn of the lateral ventricle.

The Territory of the Anterior Cerebral Artery.

One may indicate briefly the territory supplied by the anterior cerebral artery as follows:—

The whole of the mesial surface of the frontal and parietal lobes. The most posterior limit is variable; it usually reaches to the centre of the quadrate lobule, but at times it extends back as far as the parieto-occipital fissure; the most anterior limit is the post-central sulcus.

The white matter immediately subjoining the above area extending laterally into the hemisphere for a distance of about 2½ cm. A series of sagittal sections through a brain in which arteries have been injected shows that at a distance of 1 to 2 cm. from the mesial surface the most anterior wedge is supplied by the anterior cerebral artery, but there is an island in the centre corresponding with the supply of the perforating branches of the Sylvian. A section 4 cm. from the great longitudinal fissure reveals a reversal of the previous situation; the main area is supplied by the Sylvian, but an islet of anterior cerebral territory remains in the centre corresponding with the distribution of Heubner's artery.

On the orbital surface of the frontal lobe, the territory reaches laterally from the midline as far as the external limit of the internal orbital convolution.

The genu and anterior four-fifths of the corpus callosum.

The septum lucidum, the upper portion of the anterior pillars of the fornix and the medial part of the anterior commissure.

The inferior aspect of the anterior part of the head of the caudate nucleus; the anterior portion of the two outer segments of the lenticular nucleus, and the anterior half of the forelimb of the internal capsule.

Abnormalities in the Morphology of the Human Anterior Cerebral Artery.

In view of the changes which the anterior cerebral artery have undergone in the course of its acquisition of the human morphological

pattern, it is not surprising to find occasional abnormalities in its conformation. Studies of the arterial patterns in the brain, carried out in a large series of cases, have demonstrated the frequency of such deviations. Thus, in his series of 200 cases, Windle [66] found anomalies in the anterior communicating artery in 41, and in the anterior cerebral artery in 19. Fawcett and Blatchford [24] found abnormalities in the region of the anterior cerebral artery in 26 cases out of 700. It has been suggested that abnormalities of this artery occur with some frequency in the brains of the insane or amongst criminals. Wyrubow [68], for example, saw unusual forms in 22·3 per cent. of 112 brains from asylum inmates, while Parnisetti [50] found abnormalities of this artery in 34·5 per cent. cases in a series of 87 criminal brains. Amongst fœtal brains (from seven months to term) abnormalities were present, according to Lesem, in 28 out of 32 (or 87·5 per cent.).

As already mentioned, variability in mode of branching occurs relatively often, and anomalies of this order have not been included in the above statistics. Amongst these anomalies may be mentioned the emergence of certain of the frontal branches from a common trunk. Thus it is not uncommon to find the frontopolaris issuing as a branch of the anterior-internal frontal; or the anterior, medial and posterior frontal emerging from a common stem. Any combination of adjacent arteries may be encountered. Under the term "bifurcation précoce," Foix and Hillemand [27] describe the junction of the frontopolaris with the three internal-frontal arteries into a common stem which leaves the main anterior cerebral trunk at the inferior border of the frontal lobe.

The presence of one or two fine branches to the superior portion of the parietal lobe has already been noted.

Variability in the site of origin of Heubner's artery has been a common finding. Posteriorly the artery may arise as a branch of the Sylvian or of the internal carotid, while the most anterior limit is the trunk of the anterior cerebral artery, after the emergence of the anterior communicating artery.

One of the most interesting anomalies consists in the presence of a third or median anterior cerebral artery arising from the anterior communicating artery which courses upwards and backwards over the dorsum of the corpus callosum. This vessel is also known at times as the *arteria termatica* of Wilder. According to Blackburn [13] [14], it frequently divides opposite the paracentral lobule into four large equal

branches, which go to the paracentral and precuneal lobules of both sides. In such cases the anterior cerebral arteries proper are abnormally small and supply the anterior aspects of the medial surfaces of the hemispheres. The frequency with which a median anterior cerebral artery occurs is doubtful, owing to the variable statistics. Windle found it nine times out of 200 brains (4·5 per cent.); Parnisetti 7 times in a series of 87 brains of criminals (8 per cent.); Lesem 4 times in 32 fœtal brains (12·5 per cent.); Fawcett and Blatchford 23 times out of 700 brains (3·3 per cent.), and Blackburn 42 out of 400 cases (10·5 per cent.) Wyrubow reported the finding of a third anterior cerebral artery as "frequent" in his series of 112 brains from insane patients.

A much rarer anomaly is the presence of a primitive mammalian pattern, with a single azygos artery, and no anterior communicating vessel. Although Lesem found this type of artery 22 times among his 32 fœtal brains (68·75 per cent.), other observers have noted it as a rarity. It was reported by Wyrubow in his series of brains of the insane; Windle described it in 8 cases (4 per cent.).

More commonly, there may be an asymmetry in calibre between the two anterior cerebral arteries so that the vessel of one side supplies both hemispheres, sometimes by way of an hypertrophied anterior communicating artery, at other times by means of other connecting vascular channels. Or the anterior cerebral artery of one side may duplicate itself near its origin, and send one branch to the opposite hemisphere. A deformity of this type was described in 1879 by Ewart [23]. Sometimes this bifurcation occurs only after the artery has reached the dorsum of the corpus callosum (see Case 7). The first mentioned anomaly may appear, not only as a congenital morphological error, but also as the result of arterial disease. Thus, in the case of a female patient, aged 50, suffering from cerebral arteriosclerosis, the anterior cerebral artery of one side was so small in its course across the anterior perforated space as to be incapable of maintaining an adequate circulation; the opposite anterior cerebral artery therefore supplied both hemispheres by way of a large anterior communicating artery.[1]

Doubling or trebling of the anterior communicating artery is not very uncommon, and was described in 1868 by Henle [32], and eleven

[1] An almost identical phenomenon was described by Barbieri in 1867 in his fourteenth case. Heubner has given a similar illustration on p. 197 of his monograph. Other analogous cases have been reported by Rendall (1879) [51], Beaumonoir (1886) [10], and by Beadles (1907) [9] who found such a specimen in the Museum of the Royal College of Surgeons No. 3795 I).

years later by Ewart. It also occurred in 12 of Parnisetti's 87 cases, and 15 of Windle's 200 cases. Other such cases have been reported by Incoronato, Barbieri [5], Spitzka [62], Weir Mitchell [47], Godinov [28], and others. Barkow [6] [7] has described an actual vascular plexus connecting the two anterior cerebral arteries. Sometimes the anterior communicating artery is abnormal in size and calibre: thus it may be unusually short (Weber [64]; Barbieri) or unusually large (Barbieri). In one case the anterior communicating artery was found to be impervious (Barbieri). Godinov has reported that the anterior communicating artery was V-shaped in 13 per cent., N-shaped in 4 per cent., and H-shaped in 3 per cent. of his series of brains.

The anterior communicating artery is very occasionally absent; then the conformation of the anterior group of cerebral arteries recalls the primitive avian pattern (Barbieri, Spitzka). In a peculiar case quoted by Incoronato, there was no anterior communicating artery, but a circle of Willis was formed by the direct union of the two internal carotid arteries. The anterior cerebral vessels here formed a large quadrate lacuna or sinus before they separated.

Looping of the main trunk of the anterior cerebral artery is an unusual anomaly which has been reported by Shellshear in the brain of an orang-utan [58].

The Functional Significance of the Anterior Cerebral Artery.

We owe to Hilton [34] not only the conception of the constancy of arterial distribution, but also the hypothesis that the arteries are intimately associated with a definite physiological function. He quotes in illustration the internal maxillary artery as being the true "masticatory artery" in that it supplies the muscles of the lower jaw, the temporo-mandibular articulation, the pterygoid and temporal portions of the skull, and the third division of the trigeminal nerve. In recent years Shellshear [57] has applied Hilton's conception to the arteries of the brain, and he has attempted to associate the terminations of a particular vessel, not only with definite cyto-architectonic regions of the cortex as delimited by Campbell, Elliot Smith, Brodmann and others, but also with particular functional activities. The posterior cerebral artery is in this way, according to Shellshear, coupled with the mechanism of central vision, while the retinal artery is concerned with peripheral vision. It is interesting to examine the anterior cerebral artery in the light of these views.

The anterior cerebral artery, viewed phylogenetically, is intimately bound up with the development and regression of three separate structures, namely the rhinencephalon, the corpus callosum, and that portion of the motor cortex subserving leg movements.

In animals, from fishes upwards, the olfactory bulb is supplied by two vessels, known as the medial and lateral arteries of the olfactory bulb. There is in addition, commencing in the reptiles but more definitely in birds, a large ramus ethmoidalis which follows the olfactory nerve through an ethmoidal foramen. Within the nasal cavity it joins the ethmoidal branch of the internal maxillary artery to form an important plexus—the rete ethmoidalis—which supplies the mucous membrane of the nostrils and nasal cavities. These vessels are all large in calibre, though they vary in relative importance according to the species. In most mammals they outweigh the anterior cerebral arteries in magnitude, but they wane in importance with the decline in the rhinencephalon. They all arise primarily from the ramus cranialis of the cerebral carotid artery, but there are marked individual differences in the more immediate mode of origin. Thus, in fishes, the lateral and medial arteries of the olfactory bulb both arise from the carotid. In some of the reptiles and amphibians they take origin from the azygos anterior cerebral artery. The chief stem of the carotid artery in birds is continued forwards as the large ramus ethmoidalis; the anterior cerebral arteries are relatively insignificant. In mammals the ramus ethmoidalis is often called the anterior meningeal artery; it emerges at times from the anterior cerebral, but at times from the stouter marginal artery. The lateral and medial arteries of the olfactory bulb originate usually in the anterior cerebral, but also at times in the middle cerebral artery.

In man, as in the higher apes, the rhinencephalon and its vascular supply are relatively much less important. The branches of the anterior cerebral artery, particularly the prefrontal branch and at times Heubner's artery, continue to supply the olfactory bulb and peduncle and the tuberculum olfactorium. The most anterior portions of the olfactory pathways, as represented by the anterior pillars of the fornix, are also supplied by the anterior cerebral artery, but it is noteworthy that the posterior regions of the olfactory apparatus lie in the territories of other vessels. Obviously, the conception of the anterior cerebral artery as the artery of the smell-brain must be qualified: it is perhaps justifiable to regard it as the chief artery of the anterior rhinencephalon.

Prior to the development of the corpus callosum the backward

prolongations of the anterior cerebral artery are insignificant. With the specialization, however, of the dorsal commissure in monotremes and marsupials, the retro-olfactory portion of the anterior cerebral artery enlarges and is carried backward. This process advances with the phylogenetic ascent, so that as its rhinencephalic supply wanes the importance of the anterior cerebral artery as an artery of the corpus callosum increases.

The function of the anterior cerebral artery in supplying the motor cortex is always, so far as one can judge, subserved by a separate collateral branch from the main trunk. In the cat, as in most lower mammals, this vessel is an extremely fine one and may itself arise from another branch of the anterior cerebral artery. In the stag, cow and horse, however, where there is a large marginal artery, the uppermost portion of the motor cortex is supplied by one of the terminal branches of this vessel. The simian and human paracentral lobules are supplied by a branch which usually arises from the main anterior cerebral artery trunk, though at times it arises in common with the posterior internal frontal.

No satisfactory explanation has yet been forthcoming of the dual arterial supply of the motor cortex in man and apes. This mode of supply appears to challenge the views of Hilton and Shellshear. If it could be shown, however, that throughout the mammalian series the anterior cerebral artery has supplied the cortical centres supplying the limbs used for progression, and that with the development of the prehensile fore-limb the anterior cerebral artery has continued to supply the leg area only, then there would be grounds perhaps for regarding this vessel as the "artery of progression." Until such evidence is forthcoming the assignment of a physiological significance to the anterior cerebral artery will remain open.

Although there is an approximate correspondence between the territories of the cerebral vasculature and the topography of various cytoarchitectonic zones, the idea of the functional significance of the individual arteries must not be strained. Such schematization would presuppose a simplification of the anatomical basis of cerebral function which is out of accord with our conceptions of neurophysiology.

Syndromes of the Anterior Cerebral Artery.

On theoretical grounds it seems possible to distinguish between occlusions of the main trunk at various points, and thromboses of the individual branches of the anterior cerebral artery.

A.—*Occlusions of the Main Trunk.*

Here the most important differentiation has to be made between cases of thrombosis of the entire anterior cerebral artery and occlusion of the trunk after the emergence of Heubner's artery. Numerous other syndromes are theoretically possible, depending upon the site of occlusion with respect to the emergence of the various individual branches.

(1) *Total occlusion of the anterior cerebral artery (including Heubner's artery.)*

A thrombus in this position will cut off the blood supply from the whole area of distribution, namely, the mesial portions of frontal and parietal lobes and the subjacent white matter, the corresponding half of the corpus callosum, the head of the caudate and of the putamen, and the foremost half of the anterior limb of the internal capsule.

It is important to bear in mind that the thrombus must be of sufficient size to cover the point of emergence of the anterior communicating artery. If the thrombus is too short to reach this branch, the opposite anterior cerebral artery may distend the anterior communicating artery and take over the supply of both hemispheres. The result in such a case will then be almost identical with that of thrombosis of the recurrent artery of Heubner.

The chief clinical results may be stated as :—

Hemiplegia of severe degree ; some sensory loss over the paralysed lower extremity ; left-sided apraxia (if the hemiplegia is right-sided) ; mental change ; some degree of aphasia (if the lesion is left-sided).

The hemiplegia is due partly to the affection of the anterior part of the internal capsule and partly to disease of the paracentral lobule. The former will cause a severe degree of paralysis in the face, tongue and upper extremity (particularly in the proximal portion), while the latter affection will account for a marked weakness in the distal region of the lower extremity. There may be deviation of the head and eyes away from the paralysed side. The hemiplegia will usually be of the typical spastic variety and signs of pyramidal affection will be demonstrable in the increase in tendon-jerks and the presence of a Babinski response.

Softening in the cortex and subcortical white matter of the mesial portion of the post-central convolution will occasion some sensory impairment in the paralysed lower limb, particularly in its most peripheral part.

Destruction of the lateral half of the corpus callosum will interrupt the commissural fibres between the two frontal lobes and in this way is liable to produce an ideomotor type of apraxia in the left side of the

body, provided that the motor power is intact. Should the right anterior cerebral artery be affected, the left-sided apraxia will be masked by the severe motor paralysis of the upper and lower extremities; the right arm and leg will be non-paralysed and eupraxic. Owing to the escape of the splenium which is supplied by the posterior cerebral artery, a visual type of apraxia is not observed.

Some degree of intellectual loss will be present, comprising retardation, confusion and disorientation amounting at times to an actual dementia. Loss of memory may be profound, and incontinence of urine and fæces may occur. Affective changes, including emotivity, euphoria and "Witzelsucht," have not been recorded. In severer cases, all degrees of clouding of consciousness may occur, up to complete coma.

Implication of the white matter in the centrum ovale may give rise to some degree of aphasia of the executive type, often accompanied by defects in articulation. Such changes are frequently temporary.

In the following case an occlusion of the first part of the anterior cerebral artery was revealed at autopsy.

Case 1.—B.M., female, aged 95, an inmate of Tooting Bec Mental Hospital since 1925. She was admitted as a case of senile dementia of mild degree. There were no obvious abnormal physical signs in the nervous system. In the course of the next five years the mental and physical states slowly deteriorated, and she was subject to occasional attacks. During the last year of her life she showed traits of puerilism, and would nurse a rag-doll which she had snatched from another patient and thereafter refused to surrender. On March 15, 1930, she fell whilst dressing; immediately afterwards she lost consciousness and became convulsed. A condition of right-sided hemiplegia was noted, with an extensor plantar response on that side. Coma deepened and she died three days later.

At the post-mortem examination, a rupture of the heart was discovered in the region of the conus arteriosus. In the brain, a recent softening was found in the orbital and mesial aspects of the left frontal lobe: this also extended posteriorly as far as the summit of the post-central gyrus. The genu of the corpus callosum was also involved. Trans-section through the left hemisphere revealed that the softening involved the cortex and subjacent white matter of the mesial portion of the frontal and parietal lobes. The head of the caudate was entirely destroyed, and the whole of the lenticular nucleus and the anterior limb of the internal capsule were softened. On dissection of the cerebral arteries a firm thrombus was found in the left internal carotid artery, completely filling the lumen and extending along the first part of the anterior cerebral artery. The middle cerebral trunk was not occluded. There was widespread sclerosis of the cerebral vessels.

It is unfortunate that in this patient the mental state and abruptness of the stroke rendered complete neurological examination difficult. From the pathological appearance it is obvious that there had been an occlusion of the main trunk of the anterior cerebral artery at its origin; the area of softening was greater, however, than the limits of supply of that vessel, suggesting that the perforating branches of the Sylvian, or even the anterior choroidal artery, may have been simultaneously involved.

Case X in Baldy's series [4] probably exemplifies a thrombosis of the anterior cerebral artery at the point of origin:—

F., aged 67, was admitted to hospital in a state of coma which had supervened three days previously. There was a complete right hemiplegia with brachial predominance. Head and eyes were deviated to the left. Ankle clonus and a Babinski response were obtainable on the right side. Stimulation of the inner aspect of the right arm produced an abduction movement with extension of the elbow. Involuntary movements were present in the fingers of the left hand. The patient's condition was too severe to permit an investigation of sensation or of tests for aphasia and apraxia. Death occurred on the eighth day. Post-mortem examination revealed, in addition to numerous small focal lesions, a softening in the anterior and inferior regions of the anterior cerebral territory, the anterior two-thirds of the caudate and lenticular nuclei and the anterior part of the internal capsule.

(2) *Occlusion of the anterior cerebral artery trunk after the emergence of the recurrent artery of Heubner and the anterior communicating artery.*

The syndrome resulting from a thrombosis of the artery at this site is one of great interest. It comprises:—

Hemiplegia with crural predominance. Indefinite sensory impairment. Left-sided apraxia. Mental change. Some degree of aphasia if lesion is in the left hemisphere. Psychomotor phenomena in the hemiplegic upper extremity.

In this case the hemiplegia is due to the direct affection of the leg area in the paracentral lobule, with implication of the adjacent motor area. Since the only parts of the motor cortex directly within the anterior cerebral territory are the uppermost and mesial portions, which are associated with movements of the lower extremity, the paralysis will be most intense in the region of the foot and ankle, and least in the face and tongue. Although signs of pyramidal involvement are usually demonstrable, the hemiplegia is often of the flaccid variety.

Sensory changes may be found in the affected half of the body,

particularly in the lower limb, as a result of softening of the post-central gyrus.

Apraxia of the ideomotor type will be present in the left side of the body, whichever hemisphere is affected. Thus, in lesions of this type in the left anterior cerebral artery there will be a contralateral hemiparesis with a homolateral apraxia; right-sided occlusions will give ipsolateral hemiparesis with apraxia. Mental change occurs as in total occlusion of the anterior cerebral artery.

Speech defects of various types may occur with left-sided lesions. They comprise partly defects of an aphasic order, and partly those of a dysarthria; as a rule they are transient. In Baldy's case, echolalia and palilalia were also present.

Psychomotor defects in the paretic upper extremity comprise the phenomenon of forced grasping and groping (and possibly also of tonic innervation). The presence of these phenomena with the associated manifestations of "after-grasping" and "groping" can probably be associated, following Adie and Critchley's experience [1] of tumour cases, with disease of the posterior extremity of the superior frontal convolution.

Schuster, both singly and in collaboration with Pinéas, has directed attention to a phenomenon which occurred in association with forced grasping in some of his patients. On gentle stimulation of the skin in the neighbourhood of the lower lip, there resulted a pouting movement of the lips and at times a deviation of the point of the tongue towards the side of the contact (=*Ansaugen*).

The following abstracts illustrate the symptomatology of this type of arterial occlusion.

1907. *Liepmann and Maas* [41].—M., aged 70, developed a paralysis of the right leg in the course of twenty-four hours; later the right arm and right half of the face became affected and there was some defect of speech lasting a week. Mental confusion was at first present. The condition three and a half months later was as follows: Right hemiplegia, not affecting the tongue; speech a little slurred. Left apraxia and left agraphia.

1926. *Schuster and Pinéas* [56] (*Case Zeidler*).—F., aged 78, suddenly developed a left-sided weakness. Examination revealed a left hemiparesis with paralysis of the leg, no sensory loss, no aphasia, severe motor apraxia, left-sided grasping reflex and "after-grasping." "Ansaugen" was also present.

1926. *Schuster and Pinéas* (*Case Füllgraf*).—F., aged 60, right hemiparesis with maximum weakness in the leg. Tendon-jerks increased on the right side; right Babinski response. No sensory loss. Left-sided apraxia. Grasping phenomenon present in right hand.

1926. *Barré, Morin and Reys* [8].—M., aged 32: ten days after onset of a sudden headache patient had a fit. Four others followed within the next thirty-six hours. After the last attack he developed a right-sided flaccid hemiplegia with a Babinski response and died the following day.

1927. *Baldy* (*Case* 7).—F., aged 73, was examined in a state of semi-coma and found to have a right-sided flaccid hemiplegia with crural predominance. Babinski's sign was present on the right side. Sensation was apparently defective over the paralysed limb. No aphasia, but echolalia and palilalia were present. Left-sided apraxia. Memory defective but no gross intellectual enfeeblement.

1928. *Lhermitte, Schiff and Courtois* [40].—M., aged 71, developed transient weakness right arm and leg. Eleven months later he became temporarily confused and apraxic and the right leg became weaker. On examination two months later, there was found a right flaccid hemiplegia affecting chiefly the leg; ankle clonus on right, but no definite Babinski response. There was some executive and receptive aphasia. Tonic spasms appeared in the right arm resisting passive manipulations and the grasping phenomenon was present.

(3) *Occlusion of the main trunk after the emergence of the fronto-polaris and prefrontal branches.*

The syndrome following this particular lesion is illustrated by the following personal case and also by the cases recorded by Bonhoeffer, by Foix and Hillemand and by Baldy.

Case 2.[1]—E. H., female, aged 22, was admitted to the National Hospital, Queen Square, on August 12, 1895, under the care of Dr. Bastian. There was a history of a fright in November, 1894, which was followed the next day by an "hysterical fit." Two weeks later she was noticeably vacant and absent-minded and early in the morning she was found by her husband in a state of semi-coma. There had been urinary incontinence. She could not understand what was said to her. During the day she had another "hysterical fit." She was found to be helpless down the right side and aphasic, but in the course of the following month the speech returned and she regained the power in her arm. The right leg remained for a time helpless, insensitive and œdematous and then began to recover. The previous history was unimpaired except for a "fright" in 1893, following which she lost her speech for a couple of weeks and was unconscious for three hours. There had been no paralysis. On examination she was found to be deaf and aphasic. She could only just hear a loud report in each ear: comprehension of written words was perfect. Her speech was described as "gibberish." There was slight weakness of the right lower face, but the tongue was not deviated on protrusion. Motor power seemed good, but there was a distinct diminution of sensation in the right leg. The tendon-jerks were diminished in that limb.

[1] Also recorded by Mott [48] [49], with full pathological and clinical details.

After leaving the National Hospital in September, 1895, she improved till a month later when she had another fit; she was then sent to Barning Heath Asylum where she remained until February, 1896. At the time of her discharge her physical state was unchanged and she was cheerful and happy in demeanour. A year later, however, she became depressed and irritable; she frequently laughed and cried without reason; she was violent at times and had attempted suicide on one occasion. She was admitted to Claybury Asylum in March, 1897. There she was described as very emotional, irritable and depressed. She was totally deaf, but could understand written statements of a simple nature. Spontaneous speech was very sparse and badly articulated; she was dysgraphic. Physical examination revealed wasting and spasticity of the right leg without sensory impairment; bilateral ankle clonus with Babinski responses; coarse tremor of left arm.

She was transferred to Colney Hatch Asylum in June 1898: while in that institution she showed weakness of the right half of the face and tongue: both legs were spastic, especially the right. Tremor was present in right arm. Sensibility was intact but possibly delayed. She remained completely deaf. In June, 1902, she died.

Autopsy revealed, in addition to a definite mitral stenosis, three foci of softening in the brain:—

(1) In posterior third of T.1 (left) and of T.2 posteriorly, and almost all the supramarginal gyrus.

(2) A smaller area in the right hemisphere involving the posterior part of T.2, and the postero-inferior part of the supramarginal convolution.

(3) A softening in the middle portion of the mesial surface of the left hemisphere, involving most of the paracentral lobule and gyrus cingulus. The territory probably corresponded with the distribution of the anterior cerebral artery posterior to the *frontopolaris* branch.

It is difficult, of course, accurately to correlate the clinical with the pathological data, owing to the multiplicity of the foci, as well as to the indefiniteness of the clinical history. The first stroke probably corresponded with an embolus causing an infarct on the left temporal lobe; and the second attack with the lesion in the opposite hemisphere. As a result, the patient became totally deaf and aphasic. The third fit probably corresponded with the occlusion of the left anterior cerebral artery.

One is probably justified in associating the spasticity and weakness of the right leg, accompanied at one stage by œdema and by sensory impairment, with the infarcted area in the territory of the anterior cerebral artery. The mental changes are possibly connected with the damage to the left frontal lobe, though more probably the result of the multiplicity of cortical and subcortical areas of destruction.

The following case, recorded by Bonhoeffer [18] in 1914, may also be quoted in this connection :—

M., aged 51, in September, 1909, had a sudden fall without loss of consciousness ; this was followed by difficulty in speaking. The next day he fell again, this time unconscious ; on recovery was speechless. A second stroke occurred on the day following. Examination revealed a right spastic hemiplegia with no voluntary movements in leg. The tendon-jerks were brisker on the right side but there was no Babinski response. The arm gradually improved but showed later a definite catatonic tendency (*Katalepsie*), together with forced grasping. There was a bilateral apraxia, and a mixed type of aphasia.

Autopsy revealed four foci of softening :—

(1) A recent hæmorrhage in the territory of the right lenticular artery.

(2) A small softening in the left parieto-occipital lobe.

(3) A small focus in the anterior part of the internal capsule due to occlusion of the lenticulo-striate artery.

(4) An old softening in the territory of the left anterior cerebral artery with destruction of the corpus callosum as far back as the splenium, and of the anterior four-fifths of the superior frontal gyrus, and the anterior two-fifths of the median frontal convolution.

Foix and Hillemand (*Case Vig.*) (= *also Case VI. of Baldy.*).—M., aged 62, developed four years before, a weakness of her left side of sudden onset. On examination there was found a left hemiplegia affecting the foot severely ; the tendon-jerks were brisker on that side and there was a clonus and a Babinski response. Sensory changes in affected limbs. Marked intellectual enfeeblement ; no aphasia. Left ideomotor apraxia.

In Case IV of Foix and Hillemand's paper there was an occlusion of the main trunk in this situation, but the clinical examination was unfortunately incomplete. The only details available are that the patient had a left hemiplegia with contracture, affecting particularly the leg, and a left homonymous hemianopia. Forced laughter and crying were also present. The patient was admitted to hospital in a state of coma with deviation of the head to the right and with a flaccid right-sided hemiplegia.

In addition to the infarction in the territory of the right anterior cerebral artery there was a terminal softening from occlusion of the posterior cerebral artery. The left hemisphere contained a small focus in the prefrontal lobe.

(4) *Occlusion of the main trunk between the origins of the middle internal frontal and the posterior internal frontal branches.*

In the following case, recorded by Claude and Loyez [19], the area

of softening corresponds with a thrombosis of the anterior cerebral artery in this situation :—

Male, aged 51, sustained as the result of a stroke a right-sided hemiplegia with defects of speech and vision. His symptoms improved, with the exception of the weakness of the right upper extremity. Five months later a second stroke was followed by a sensation of burning in the left upper limb, and there were vasomotor changes and cyanosis. Examination showed a condition of right-sided hemiparesis with brachial predominance ; tendon-jerks were brisker on the right side ; both plantar responses were flexor in type. There was no gross defect of sensation, but there were errors of topognosis and of tactile discrimination equally pronounced over both halves of the body. There was a left-sided apraxia of mixed ideatory and ideomotor type. Psychical changes included defect of memory, inability to perform mental calculations and inability to read or write. There was no aphasia, but the speech was somewhat stammering.

Three years later a fresh stroke occurred and was followed by the appearance of pseudo-bulbar signs. Death occurred two years afterwards.

(5) *Occlusion of the main trunk between the origin of the posterior internal frontal and paracentral arteries.*

This particular type of lesion is probably exemplified in the case recorded by Goldstein [29], [30] :—

A female, aged 57, developed a left-sided hemiplegia which improved, but left a severe permanent weakness in the leg. Synchronously with the return of strength certain disorders of mobility made their appearance. Thus, in addition to a unilateral apraxia with marked poverty of spontaneous movement, the phenomenon of tonic innervation was present. Amorphous movements of a "pseudo-spontaneous character" made their appearance when she voluntarily attempted an action. She was unable to write with the left hand.

B.—*Occlusion of Individual Branches of the Anterior Cerebral Artery.*

Unfortunately our knowledge as to the clinical effects of thrombosis of the various branches of the anterior cerebral artery is still very incomplete, but a few records are quoted to illustrate one or two such instances.

(1) *Occlusion of Heubner's artery.*

This was present in one personal case in which there were actually two thromboses in the anterior cerebral branches. There was an exact correspondence in the size and position of one infarcted area with the territory of this vessel, and the shrunken and occluded artery of Heubner was easily demonstrable.

Case 3.—C. W., male, aged 57, was admitted to the National Hospital, Queen Square, under the care of Dr. F. M. R. Walshe on November 8, 1928. Five months previously he had experienced a sudden loss of speech, accompanied by a slight weakness of the right arm. This became worse two days later. Three weeks afterwards, a weakness was noticed in the right leg. For the three months immediately prior to admission his speech as well as the paresis of the leg had been improving, but the arm remained completely helpless. Some psychical change, such as irritability and forgetfulness, had been noticeable.

He was a stout florid man with complete inability to speak; he could understand and execute simple commands, however, but could not read. There was no apraxia. He showed some emotional incontinence.

There was possibly a right-sided homonymous hemianopia. The right pupil was larger than the left. Slight weakness was present in the right half of the face, palate and tongue.

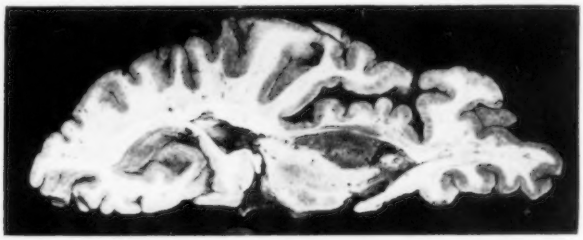

FIG. 12.—The region of the basal ganglia of the left hemisphere from *Case* 3. There is a softening in the head of the putamen and caudate nucleus, exactly corresponding with the distribution of Heubner's artery.

The right arm was spastic and no voluntary movements were possible with the exception of a few feeble movements in the fingers. When the arm was held away from the trunk a rhythmical tremor was evident. There was some slight weakness of the right leg with increase of tonus. Sensation was apparently intact. The abdominal responses were absent and both plantars were of the extensor variety. Tendon-jerks were brisker on the right.

Blood-pressure measured 210/110; the radial and retinal arteries were distinctly sclerotic. The heart sounds were tic-tac in character and the second aortic sound was ringing. Blood Wassermann reaction was negative.

The patient died on December 2, 1928. Autopsy revealed three foci of cerebral softening:—

(1) A large focus in the left hemisphere in the territory of the middle and posterior internal frontal branches of the anterior cerebral artery.

(2) An old small softening immediately beneath the cortex of the left angular gyrus, involving the optic radiations.

(3) A softening in the head of the putamen and caudate nucleus on the left side. The anterior half of the fore-limb of the internal capsule was also involved. (See fig. 12.)

In this case it is likely that the third-mentioned lesion, due to the occlusion of Heubner's artery, corresponded with the first stroke, while the weakness of the right leg, appearing three weeks later, is to be associated with the thrombosis of the middle and posterior internal frontal branches. If this is so, then the severe degree of weakness of the right upper extremity—involving the whole limb with conservation of feeble mobility in the fingers—the spasticity and the tremor are referable to this lesion. The slighter degree of paralysis of face, palate and tongue are also connected in all probability with this particular focus. It is probable that the aphasia is in part referable to the lesion of Heubner's artery, though alexia is clearly due to the affection of the angular gyrus.

(2) *Occlusion of the frontopolaris branch.*

There is no clinical record of a softening confined to the territory of the frontopolaris branch, though Foix and Hillemand have given an illustration in their paper of the brain of such a case.

(3) *Occlusion of the middle and posterior internal frontal branches of the anterior cerebral artery.*

The symptoms of this type of thrombosis are exemplified by the ollowing three cases:—

Case 4.—F., aged 33, was admitted under the care of Dr. J. Odery Symes to the Bristol General Hospital on August 5, 1924, in a state of coma. The history was that she had been confined twelve days previously: no abnormality had been noted until 2 a.m. of the twelfth day, when she said she could not move her body although she was actually able to move her limbs and appeared perfectly sensible. At 7 a.m. she began shaking violently and half an hour later she lost consciousness. When admitted at 2 p.m. she was still unconscious; respirations were stertorous. She became "rigid and shaky" when handled. Pulse rate 152. Respiratory rate 38. Temperature 101° F. Blood-pressure 130. Urine normal.

During the course of examination she went into a generalized fit. There was no pupillary abnormality: the knee-jerks were present on both sides, although they disappeared later the same day. A bilateral Babinski response was obtained. Several other fits supervened during which the right half of the body was rigid and immobile, while the left arm and, to a lesser extent the left leg, twitched. Fits followed in rapid succession, and the unconsciousness became more profound. The march of the attack became more definite, commencing in the eyelids and face and proceeding thence to the left arm and finishing in the hand; no other limb became convulsed. Head and eyes were deviated to the left. The cerebrospinal fluid was not under obvious tension. It was yellowish in tint and slightly turbid; on standing a spider-web clot formed. Total protein 0·3 per cent: globulin in excess; cell count 350 per

cmm., of which 85 per cent. were polynuclears. Culture sterile. The temperature rose to 107° F. the next day, the respirations became more laboured and death occurred forty-eight hours after the onset of cerebral symptoms.

Autopsy revealed an infarct in the region of the middle and posterior branches of the left anterior cerebral artery.

Case 5.—J. W., male, aged 42, was admitted to the National Hospital, Queen Square, on May 26, 1924, under the care of Dr. J. L. Birley, suffering from Jacksonian attacks and weakness in the left leg. The history dated from 1916: Whilst a soldier on parade he was observed to stagger and was sent to the guard room as drunk. Whilst under arrest he had a fit which was followed by paralysis of the left side of the body and some affection of speech. In the course of a few weeks the speech defect cleared up and strength returned to the arm. Later the leg regained power. The following year another fit occurred but left no sequelæ. Thereafter other attacks followed in great

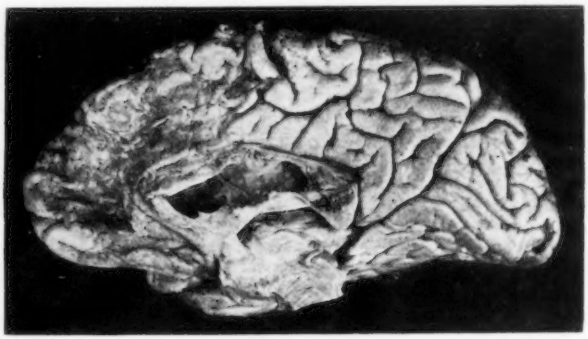

FIG. 13.—Large focus of softening in the right hemisphere from *Case* 5, corresponding with the distribution of the middle and posterior internal frontal branches of the anterior cerebral artery.

number. There were two chief types of fit: (*a*) attacks consisting in a "funny feeling in the head," together with twitching of the left arm and knee, without loss of consciousness, and (*b*) major fits with unconsciousness.

Previous health was uneventful except for syphilis contracted at age of 18.

His physical condition on admission was as follows:—

Optic discs somewhat blurred; nystagmus on lateral ocular deviation; clumsiness and dysdiadochokinesis of the left upper extremity. The left leg was spastic and weak. There was an indefinite blunting to pin prick over the left hand. The tendon-jerks were brisker on the left and there was a left Babinski response. The diagnosis was made of a right-sided tumour in the motor area, but an exploratory operation failed to reveal any definite abnormality.

For a year following the decompression he was free from fits. They then returned and his left leg became weaker so as to interfere with his walking.

He was re-admitted to the hospital on September 23, 1929, under the care of Dr. J. P. Martin. He now showed definite psychical changes, especially gross retardation. His expression was vacant.

The disc edges were still hazy. The pupils were now Argyll-Robertson in type. Both hands were tremulous and somewhat ataxic; there was dysdiadochokinesis in the left arm, and the right grasp was definitely stronger. The left leg was weak and spastic and lay in an attitude of extension. The reflexes were brisker on the left side, and both plantar responses were flexor in type. No sensory change was demonstrable.

The diagnosis was made of dementia paralytica and the patient died on October 28, 1929. Autopsy revealed the macroscopic changes of general paralysis of the insane which were substantiated by histological examination. In addition there was an old softening in the territory of the anterior cerebral artery and the middle internal frontal was reduced to a fine impervious thread. The posterior internal frontal, which arose as a branch of the middle internal frontal artery, was similarly affected. The main trunk of the anterior cerebral artery was not occluded. (See fig. 13).

In Case 3 it will be remembered that three softenings were demonstrated at an autopsy; one lay in the angular gyrus, and one in the basal ganglia in the territory of the artery of Heubner. The third focus occurred in the area supplied by the middle and posterior internal frontal arteries (see fig. 14). The sudden attack of weakness in the right leg occurring three weeks after the original attack can probably be referred to this particular lesion. We may, therefore, refer to this as the third example of occlusion of the middle and posterior internal frontal vessels.

Of these three cases the first is obscured by the severity of the general symptoms which excluded an intimate examination. The two most important focal manifestations were left-sided convulsions and deviation of the head and eyes towards the left. In the light of the pathological evidence this can be correlated with the left-sided cerebral infarction which, from the suddenness of its onset, must have caused a severe degree of right-sided hemiplegia and deviation of head and eyes to the opposite side. The non-affected right half of the body, therefore, showed the only involuntary movements in what were otherwise generalized epileptiform convulsions.

In the second case the most obvious findings consisted in a spastic crural monoplegia with an extensor plantar response, together with some degree of awkwardness of the ipsolateral upper limb. The occurrence of local seizures commencing in the left hand were here probably in the nature of irritative Jacksonian fits, and were frequently unaccompanied by loss of consciousness.

The third example (i.e., Case 3) is complicated by the plurality of lesions, but one is probably justified in referring the spasticity and weakness of the right leg to this particular focus.

(4) *Occlusion of the paracentral artery.*

Here the area of ischæmia involves the cortex and subjacent white matter of the paracentral lobule including that portion of the motor cortex associated with the innervation of the foot and lower leg. This lesion has been verified in the cases recorded by Long (1907) [42] [43], Wilson (1923) [65], and by Winkelman (1924) [67].

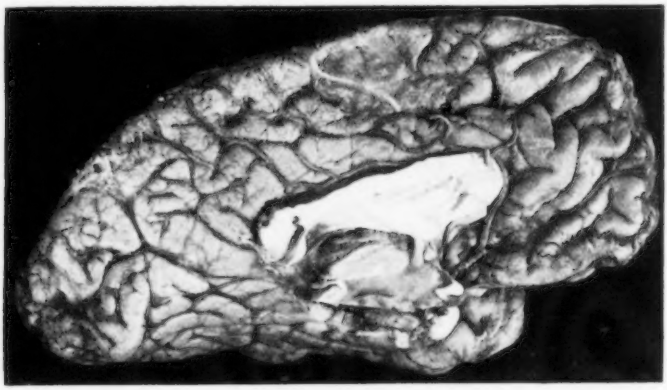

FIG. 14.—Brain from *Case* 3, showing a softening in the area of supply of the middle and posterior branches of the left anterior cerebral artery.

In Long's patient, a man of 72, there was a history of a sudden weakness in the left leg: the arm and the face were not affected. The movements in the leg were found to be limited in range and feeble in strength, the foot lay in an equinovarus attitude. The knee-jerk was exaggerated on that side, and although the plantar response was extensor in type, there was no ankle-clonus. Five years later the physical signs were unchanged. The site of the lesion was determined at autopsy.

Wilson's Case 1 was that of a woman aged 74, who three weeks previously had developed a sudden weakness of the right foot. In a few hours this spread to the leg and increased in intensity. On examination, the leg was œdematous and in a position of eversion. No voluntary movement was possible: passive manipulations evoked pain. There was slight clonus at the right ankle and the plantar response on that side was of the extensor variety. Sensation was normal. Autopsy revealed a softening of the paracentral lobule and uppermost tip of the motor cortex.

Winkelman's Case 2 concerned a coloured woman aged 38 who developed a

sudden paralysis of the left leg with numbness and tingling. Six months later she died from a second stroke. At autopsy, an old softening was found in the paracentral lobule, in addition to a recent thrombosis in the internal capsule.

The outstanding clinical feature in the last group of cases has been a weakness of the contralateral lower limb. In some instances it exists alone in a crural monoplegia, while at other times it accompanies a lesser degree of weakness of the arm, face and tongue on the same side. The maximum weakness is found in the distal portion of the leg. In most instances the affected limb is spastic, with exaltation of the tendon-jerks, ankle-clonus, and an extensor plantar response. In not a few the limb was markedly œdematous; sensory changes are also present at times.

On looking over the clinical results of thrombosis of the anterior cerebral artery at various points and of occlusion of its various branches, certain symptoms stand out by reason of their frequency. Three symptoms in particular attract attention, namely, paralysis of the lower extremity, psychomotor disorders in the ipsolateral upper limb, and ideomotor apraxia affecting the left arm, whether that limb be ipsolateral or contralateral. The characteristics of the crural monoplegia have already been noted. This particular symptom is the most striking of the manifestations of disease of the anterior cerebral artery, and only in the case of thrombosis of the main trunk between the origin and the anterior communicating vessel is the monoplegia masked by the addition of a severe capsular type of hemiplegia. Since the paracentral lobule represents the only area in the territory of the anterior cerebral artery which possesses an obvious and localized clinical significance, the explanation of the frequency of crural monoplegia is obvious.

Cases of paralysis of one leg arising suddenly in the course of a cerebral arteriosclerosis or cases of hemiplegia with crural predominance are not very uncommon. In the course of the last three years seventeen such cases have come under my notice. It is rare, however, for pathological verification to be forthcoming in such cases as the causative lesion is rarely sufficiently severe as to cause death.

Lesions of the cerebral arteries, other than the anterior cerebral, are very unlikely to cause a marked paralysis of one lower extremity without affecting equally severely the arm, face or tongue on that side. The anterior cerebral artery, however, by reason of its exclusive supply of the leg area—which receives no blood from other sources—characteristically produces, when diseased, an affection of the lower

extremity. It is highly probable that the great majority of cases of crural monoplegia of sudden onset, occurring in the course of a cerebral arteriosclerosis, are due to lesions of the anterior cerebral artery or its branches.

There is one particular vascular lesion, however, which closely resembles in its clinical features occlusion of the paracentral artery—namely, thrombosis of the vein of Rolando. In Waggoner's case [63] a man of 76 awoke one morning to find his right leg paralysed. The arm was also slightly weak but rapidly regained its strength. On examination, the right leg was found to be weak and the whole right side was incoordinate. Tendon-jerks were brisker on the right, the abdominal reflexes were absent on that side but there was no Babinski response. The only sensory loss consisted in an astereognosis of the right hand. At autopsy a large thrombosis of the left Rolandic vein was found overlying a plexiform hæmangeioma.

Closely analogous with the foregoing are the cases of injuries of the superior longitudinal sinus, resulting from shrapnel or gunshot wounds of the vertex of the skull, as described by Holmes and Sargent [36]. Whilst in the majority of cases symptoms of bilateral and symmetrical distribution were the result, sometimes one side of the body was affected alone: this was the result of an injury directed to one side of the middle line, blocking the circulation through a lateral lacuna or in the veins as they enter the sinus. Of their series of twenty patients, five showed symptoms affecting one leg only. The clinical results of such a lesion comprised a paresis of the lower extremity most marked in the distal segments: severe rigidity with exaggeration of the tendon-jerks and extensor plantar responses; a cortical type of sensory loss, as shown by gross defect of postural sensibility and tactile discrimination, with little or no impairment to light-touch, pain or temperature. The attitude of the paralysed limb was usually one of extension with adduction and internal rotation; the big-toe was often maintained in the so-called "Babinski attitude."

Ipsolateral psychomotor disorders are important in most cases of thrombosis of the anterior cerebral artery; they are absent from cases of thrombosis confined to the paracentral branch. Although probably of frequent occurrence, it is possible that at times they are overlooked, particularly when mental change or obfuscation of consciousness complicates the picture. Characteristically, they comprise the phenomena of forced grasping and groping. In Goldstein's case tonic innervation

was present, probably in addition to a grasp reflex. Other psychomotor anomalies include the presence of catatonia and the "pouting" and "sucking" reflexes noted in Schuster's patients. Goldstein's patient also showed a distinct hypokinesia and an absence of "movement-intention." Voluntary movements were accompanied by the appearance of amorphous, pseudo-spontaneous movements, and there was a severe disturbance of optical imitation.

Softening of the corpus callosum occurs in many of the types of occlusion of the main arterial trunk, and gives rise to the appearance of apraxia in the left side. The apraxia is of the ideomotor variety, and is shown by the patient's inability to carry out a movement to command, such as saluting, waving the hand or blowing a kiss. The apraxia is always unilateral and left-sided, since, according to Liepmann's conception, the centre controlling eupraxia is situated in the left supra-marginal region and is associated with the motor centres of both sides. The connections between the left supra-marginal gyrus and the right motor cortex pass by way of the corpus callosum. Damage to this commissure will, therefore, result in left-sided apraxia.

When the thrombus lies in the right anterior cerebral trunk at the point of emergence of Heubner's artery, the resulting severe degree of hemiplegia will obscure the apraxia.

Bilateral Lesions.

The presence of multiple foci in a case of cerebral arteriosclerosis so obscures the symptomatology that localization of the individual lesions becomes almost impossible. This is particularly true when the softenings lie in the territories of different cerebral arteries, such as the posterior cerebral artery on the one side and the Sylvian of the other. In the case of the anterior cerebral artery, however, it would seem theoretically possible to diagnose a bilateral symmetrical lesion, on account of the peculiar distribution of motor weakness. Thus a sudden weakness of one leg, followed later by a sudden weakness of the other, together with spasticity and signs of pyramidal defect, and little or no sensory change, would be suggestive of a bilateral affection of the anterior cerebral arteries. It is possible that some of the cases of so-called "senile paraplegia" belong to this group.

Case 6 illustrates the occurrence of bilateral, symmetrical softenings situated within the territory of branches of the anterior cerebral artery.

M. J., female, aged 88, an inmate of Tooting Bec Mental Hospital since 1913. She was admitted as a case of secondary dementia, and there was no

gross abnormality found in the nervous system. Marked arteriosclerosis was present. During the seventeen years she was under observation she gradually deteriorated; there was at no time any definite illness except for occasional appearance of œdema and albuminuria. There was never any evidence of a stroke. She steadily became weaker and died in January, 1930, of heart failure following bronchitis.

At the autopsy there was found an old infarction of the left ventricle of the heart. The cerebral blood-vessels were grossly degenerated. There was a bilateral softening lying in the orbital aspect of both frontal lobes. The area was a little larger in the left hemisphere where it involved the pole of the frontal lobe as well as the gyrus rectus. On the right side the gyrus rectus alone was destroyed. An old porencephaly was present at the apex of the left temporal pole.

The softenings evidently lay within the territory of the prefrontal branch on the right side, and of the prefrontal and frontopolaris vessels on the left.

In the following case, a bilateral lesion in the territory of the anterior cerebral arteries produced a spastic paraplegia of cerebral type. The syndrome resulted, however, from a single lesion on account of a congenital peculiarity in the arterial distribution whereby the left anterior cerebral artery supplied both hemispheres:—

Case 7.—S. P., male, aged 45, was admitted to the National Hospital, Queen Square, under the care of Dr. J. L. Collier on November 26, 1920. Eleven months previously, on being informed that he had lost all his savings, his legs became immediately stiff and tremulous, and there was great difficulty in walking. His symptoms improved somewhat after three weeks, but became worse again five weeks before admission. There was some slight difficulty in micturition but no other sphincter disorder. Since the onset of his symptoms he had become impotent. There had been some psychical change within the past year; his memory was not so good, he was slower in his actions and his employers noticed that his work had fallen off so that he could no longer use a micrometer.

On examination, the right pupil was found to be larger than the left, but both reacted normally; there was a slight bilateral ptosis, and a few ill-sustained nystagmoid jerkings on lateral deviation of the eyes. The other cranial nerves were normal. There was no loss of motor power in the arms, but the tonus was somewhat increased and there was a distinct catatonic tendency. Both lower limbs were very spastic and the right leg was extremely weak, especially in the distal segments. There was no sensory loss other than a defect of postural sensibility in his toes on the right foot. The tendon-jerks were exaggerated on both sides and there was a left-sided ankle-clonus. The abdominal and cremasteric responses were present and both plantar reflexes were extensor in type. The cerebrospinal fluid was normal. Blood-pressure was 240.

Progress notes: On December 6, 1920, he suddenly cried out with intense

headache, vomited and then lost consciousness. Coma deepened the next day and respirations became Cheyne-Stokes in type. A definite spasticity was now noticeable in the arms; a second lumbar puncture revealed a blood-stained fluid. He died on December 12, 1920.

Autopsy revealed a marked degree of sclerosis of the cerebral blood-vessels. There was a quantity of blood free in the subdural space. More careful examination of the cerebral arteries showed that the right anterior cerebral artery was extremely small in calibre and supplied only the most anterior portion of the frontal pole. At the level of the genu the left anterior cerebral artery, which was of greater size than the right, bifurcated and sent one branch across the middle line so as to supply the mesial aspect of the right hemisphere. In this way the left anterior cerebral artery was supplying the cortex, including the paracentral lobules, of both halves of the brain. A thrombosis of the first part of the main trunk of the left anterior cerebral artery was present, causing an ischæmia of the cortical areas corresponding with both lower limbs.

A small aneurysm was present on the right middle cerebral artery, as it lay in the Sylvian fissure.

Weigert-Pal preparations of the medulla and spinal cord showed a bilateral degeneration of the pyramidal tracts equal in degree on the two sides.

Cases of bilateral paralysis due to verified lesions of both anterior cerebral arteries are found in Baldy's fifteenth case and Schuster's [54, 55] case Warkow. The former was a woman who had a left hemiplegia with crural predominance, and left-sided hemianopia and forced laughter and crying. She was admitted to hospital on account of a sudden right-sided hemiplegia of flaccid type. Full examination was not possible as she was comatose. At autopsy three softenings were found; one in the territory of the right anterior cerebral artery, a cuneiform focus in the mesial portion of the left prefrontal lobe, and a recent softening in the territory of the right posterior cerebral artery.

Schuster's patient (Warkow) was an old man, aged 73, who had two strokes within four years. As a result he became paralysed in both legs and demented. He made no spontaneous utterance, and was disoriented in space and time. The right corner of the mouth was weak; the right hand moved restlessly in different attitudes. The left forearm was held tightly across the chest with the fist clenched. With the right hand the patient clutched the bedclothes firmly or else grasped neighbouring objects, such as the examiner's pencil, stethoscope, &c., and force had to be used to make him release the objects. Both legs were weak and there was an increase of tonus on the left side. The tendon-jerks were increased, especially on the left; the plantar responses were in extension. At post-mortem examination there was a bilateral softening of the anterior cerebral arteries.

The cases of bilateral symmetrical foci of softening in the prefrontal lobes recorded by Zacher [69], and by Mabilles and Pitres [44], may also be examples, but it is difficult to be certain from their pathological descriptions which particular vessels were occluded.

In addition to these instances of abrupt bilateral lesions of the anterior cerebral arteries, it is possible that there may also occur progressive affections of the two arterial trunks, so as to cause gradual bilateral manifestations of a paraplegic order. Thus in Marie and Foix's case [46] of cerebral paraplegia from progressive subependymal necrosis, a condition of paraplegia in flexion, dementia, dysarthria and emotional incontinence developed in a woman of 70. The pathological process consisted in an atrophy of the corpus callosum, and a progressive softening of the upper group of pyramidal fibres in their transition from the paracentral lobule through the white matter. Analogous cases have also been reported by van Bogaert and Ley (1926) [15].

Finally, one may mention briefly the condition of progressive degeneration and gliosis of the corpus callosum described by Marchiafava [45] in alcoholic subjects. Although this process is usually regarded as due to toxic action of the alcohol, Bonfiglio [17] has pointed out a local sclerosis of the neighbouring blood-vessels, and has suggested that the process is an ischæmic one, due to a progressive affection of the branches of the anterior cerebral artery.

Aneurysm of the Anterior Cerebral Artery and its Branches.

The anterior cerebral artery is one of the less common vessels to be the seat of aneurysm formation. Thus Peacock found aneurysms in the anterior cerebral or anterior communicating arteries in 9 out of 89 cases; in Krey's series of 142 cases an aneurysm occurred in the anterior cerebral artery in 11; Gowers found aneurysms on the anterior cerebral artery 14 times, and on the anterior communicating artery 8 times in his 154 cases. Van Hoffmann recorded 78 new cases of intracranial aneurysm, and placed the anterior communicating third and the anterior cerebral artery sixth or last in his list of common sites. Fearnsides [26] found in his 51 cases that the aneurysms arise from either the anterior cerebral artery or the anterior communicating artery in 15 cases.

In an attempt to analyse the cases of aneurysm of these vessels only those instances will be studied in which the site of the lesion was clearly defined at autopsy. The symptomatology then resolves itself into two main groups: first, the clinical evidences due to the size of the

aneurysm and its contact with neighbouring structures; and secondly, the results of rupture or leakage.

In the great majority of cases there have been no focal or general signs of localizing value, and the demonstration of an aneurysm at autopsy has in many instances been unsuspected. Amongst the focal signs, affections of the first and second cranial nerves on one side are important. There results a unilateral anosmia and a progressive dimness of vision in one eye, advancing to complete blindness. The defect in the visual fields comprises a progressive scotoma which ultimately fuses with an incoming peripheral loss. Ophthalmoscopy reveals optic atrophy. Depression, and later abolition, of the pupillary reflex to direct light is found in the affected eye, although the consensual light response is maintained. In one case of Fearnside's the blind eye was totally anæsthetic.

When the aneurysm arises from the anterior communicating artery, pressure may be exerted upon the optic chiasma so as to produce ultimately a bitemporal hemianopic defect. This occurred in an interesting case of Weir Mitchell's [47] where the tumour grew from an anomalous vessel connecting the two internal carotid arteries, and which probably represented a doubling of the anterior communicating artery. The presence of other so-called "pituitary signs" usually indicates an aneurysm arising from the internal carotid.

Diplopia, without an obvious ocular palsy, may also result from direct pressure upon oculomotor nerves.

Erosion of the sella turcica or other portions of the floor of the skull may be demonstrable by X-rays, and there may also be revealed incomplete ring-like suprasellar shadows in cases of calcification of the aneurysmal sac.

Although in most cases no bruit is audible on auscultation of the skull, the patient may nevertheless complain of tinnitus. An insane patient of Beadle's, in whom an aneurysm of the anterior cerebral artery was afterwards demonstrated, complained for many years of a "waterfall inside the head."

It is noteworthy that in no confirmed case was there any clinical evidence of occlusion or other sign of interference with the functional activity of the anterior cerebral artery.

In addition to the signs of local pressure which are occasionally demonstrable, certain general manifestations may occur as with aneurysm elsewhere. Thus, paroxysms of headache are not uncommon, while drowsiness may also be observed. Papilloedema occasionally occurs and the

combination of swelling of the optic disc in one eye, and atrophy in the other, is a particularly suggestive phenomenon. Mental changes may be present in a few recorded cases, but it is very doubtful to what extent these are directedly connected with the presence of the aneurysm.

The clinical manifestations of rupture or leakage of blood from the aneurysm are very similar wherever the site of origin of the lesion. It is rarely possible to predict from the clinical signs that the aneurysm lies on the anterior cerebral artery. In some cases there have arisen signs of a mild paretic order on the side of the body opposite to the blind eye. Ptosis has been observed in a few cases, sometimes bilateral, but at times in the opposite side only; paralyses of ocular movements may occur, but they are not of localizing importance. Dilatation and reflex immobility of the pupil on the side of the aneurysm may occur, but has also been described in the opposite eye. In all cases of rupture of aneurysms growing from the anterior portion of the circle of Willis, hæmorrhage may track along the optic nerve-sheaths, but this sign cannot be regarded as diagnostic of aneurysm of the anterior cerebral artery.

In our present state of knowledge, therefore, it is doubtful whether it is possible with any confidence either to lateralize or to localize an aneurysm of the circle of Willis. This holds true for the case of both the ruptured and unruptured aneurysm. Although it is frequently possible to predict whether the aneurysm lies in the anterior or the posterior portion of the circle of Willis, it is not possible to predict with certainty whether the internal carotid, the Sylvian, the anterior cerebral or the anterior communicating artery is the region affected.

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A THEORETICAL APPLICATION TO SOME NEUROLOGICAL PROBLEMS OF THE PROPERTIES OF EXCITATION WAVES WHICH MOVE IN CLOSED CIRCUITS.

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THE phenomena of spontaneous involuntary movements have never been satisfactorily explained. For many years neurologists have attempted to understand them solely in terms of Hughlings Jackson's conception of the release of a "lower" centre from the regulative or inhibitory control of a "higher" one. It cannot be claimed that these efforts have been very successful. Indeed, the absence of adequate pathological or experimental data to support this view is particularly striking when one recalls how fully the doctrine has been worked out by Sherrington and his school for the functional relationship of the brain-stem and cord to higher levels. This sharp contrast between the fruitful use of the conception in one direction, and its futile application in another, leads one to suspect that the phenomena involved may be fundamentally different.

There is, in fact, one quite obvious difference between spontaneous involuntary movements on the one hand, and, on the other, the experimentally investigated manifestations of inhibition and release. The former are purely pathological processes; the latter are all components of normal function, e.g., postural tonus, fragments of normal movement patterns, and myotatic reflexes. In the experimental study of these mechanisms, something normal may be exaggerated (i.e., "released from inhibition"); or, by appropriate stimuli, some normal response may be decreased (i.e., "inhibited"); but no chaotic patterns of new movements are produced. If, therefore, one attempts to explain the presence or absence of spontaneous involuntary movements by processes of release and inhibition, it is their actual *creation* by release and their *total extermination* by inhibition which must be accounted for. This is not merely a normal mechanism undergoing increase or diminution; it presupposes a process of inhibition and release which actually goes beyond experimental demonstration, and which leaves untouched the problem of what mechanism it is which is being released or inhibited.

Secondly, it must be emphasized that afferent stimuli produce postural tone and myotatic reflexes, not merely under the abnormal conditions of decerebration, but normally as well. On the other hand, it has not yet been proved that spontaneous involuntary movements depend in any way upon an influx of afferent stimuli. But even if it were shown that the elimination of all afferent impulses abolished such movements, it would be justifiable to conclude only that, without afferent impulses, no spontaneous movements can occur. It would remain obvious that since afferent stimuli are always occurring normally, and since spontaneous involuntary movements do not occur normally, therefore the afferent stimuli alone are not the essential mechanism although they may be a *sine qua non* of the phenomena.

In short, these are two different kinds of phenomena; one of them normal but subject to pathological exaggeration or diminution, the other found only in abnormal states; one of them directly produced by proprioceptive stimuli, the other dependent upon afferent impulses under abnormal conditions only, if at all. Obviously what may be an adequate explanation of the quantitative variations of the one can only play at most a contributory rôle in producing the other.

Out of the manifold possibilities with which the central nervous system is endowed, an attempt is made here to see if these obscure "spontaneous" neurological phenomena become any more understandable if one simply pictures the central nervous system as a place in which, under certain conditions and in certain areas, excitation waves move along pathways which ultimately return them to their starting points. This possibility is selected because such a circular wave would constitute a source of energy, which under certain conditions would give little or no outward signs of its existence, but which, with a slight change in conditions, might suddenly become manifest. That the nervous system is a "storehouse" of energy in some form is an old and necessary conception. The tacit assumption has been that this is some unknown form of static energy which can on occasion be transformed into nervous impulses and "released." The speculations presented here suggest that these phenomena can all be understood in terms purely of excitation waves and axonal impulses.

This implies that the excitation wave and the axonal impulse are not necessarily identical, a position which Sherrington has also found useful and necessary, and which he has expressed in his surmises about an "excitatory agent." Furthermore, it challenges a too rigid adherence to the neurone doctrine—denying that confidence in the general validity

of the doctrine which imposes upon us the belief that no disturbance to the polarity of the neurone or synapse can ever occur, even in abnormal states.

In order to understand how excitation waves which run in closed circuits can affect the surrounding tissues, it is necessary to review their rather peculiar properties. Such a circular wave constitutes a delicately balanced system whose course can be changed, and indeed whose very existence can be terminated by slight changes in the inter-relations of four sensitive factors: (1) the initial intensity of the wave (which for any single pathway in which the All-or-None principle rules will depend upon the state of conductivity of the tissue); (2) the velocity of the wave; (3) the length of the circuit; (4) the duration of the relative or absolute refractory phase which follows the passage of the wave. For instance, if some metabolic change or an alteration in blood-supply or an appropriate drug should lengthen the duration of the absolute refractory period, the wave on its next circuit might reach tissue which was still refractory and through which it could not pass; and thus it would be quite annihilated. The same thing might result from a change in velocity. Or a combination of such changes, even without any alteration in the anatomical organization of the tissue, might simultaneously close a normal pathway and open an unusual one, thus altering the direction and the length of the circuit through which the wave had to pass. And by similar changes not only may closed circuits be converted into open ones, but open circuits may be thrown into new relationships and closed circuits set up. If, therefore, it should prove possible to find any good reason to believe that circular excitation waves exist normally in any parts of the nervous system, it would be possible to use such waves in explaining spontaneous neurological phenomena.

(It may be worth while to recall that just such a system as this is at present supposed by Sir Thomas Lewis and others to provide the mechanism of auricular flutter and fibrillation, the former being due apparently to a stable circus movement over a constant course, and the latter to movements over irregular and varying circuits.)

Where, then, in the nervous system might one look for such pathways? In answering this question it becomes necessary to give to the loosely-used concepts of "active areas" and "silent areas" a quite precise meaning. By "active areas" will be meant all of those structures over which no nervous impulse can pass without producing some bodily change; whereas by "silent areas" will be meant those tissues through which an impulse may pass without reacting upon the rest of the body.

In thinking about the central nervous system, it is customary to assume that all impulses or excitation waves move from some point of origin to some terminus, over pathways all of which lead either INTO or ultimately OUT OF the central nervous system. However complicated such paths may be in detail, all of them are essentially open circuits, like a horse-shoe—with an afferent and an efferent limb. Let us accept this as a true picture for all of those parts of the brain which we ordinarily designate as "active,"—i.e., those which have a direct motor, sensory, or vegetative significance Roughly speaking, then, throughout this "active" tissue there can be no play of impulses without either visible movement or sensible feeling or some alteration of internal economy. When the body is at rest, the tissues of these active areas of the brain must be at rest. (It is not essential to this reasoning to discuss whether this state of rest is absolute or merely relative, i.e., below a threshold value.)

Within the so-called "silent" areas of the brain, on the contrary, it is possible to imagine a state of constant activity, even when the body is at rest. In fact, it is fairly clear that not only is this possible, but it would seem to be a very probable state of affairs. Furthermore, this activity can be quite plausibly pictured as consisting of innumerable excitation waves circling continually in closed and silent pathways.

The appeal of this concept rests upon the following reasons :—

At every moment the central nervous system is receiving proprioceptive and exteroceptive stimuli in great numbers. To these impulses one of three things may happen : they may pursue an unchecked course from afferent to efferent pathways (i.e., the horse-shoe circuits of the active areas of the brain) ; or they may enter refractory areas in which they are destroyed ; or they may encounter refractory pathways in the active area, but pathways that are open into the silent areas. Once within the silent areas they must either circle until they are destroyed, or ultimately come out again into active efferent expression. On some such basis one may picture the circuits of the silent areas arising. It is equally possible, of course, to postulate the existence of centres which spontaneously generate and discharge impulses within these silent areas. But such a postulate is merely another speculation, and the impulses so generated would also have to meet with one of these three fates. So that it seems to lessen the speculations by at least one hypothesis, to think of the activity in the silent areas as being the result of afferent impulses diverted from the active afferent system into the silent areas and circling there. But whatever their hypothetical origin, the im-

portant thing to emphasize is that impulses playing about within the nervous system face one of three possible fates: they must either go in open circuits which must ultimately lead to efferent expression; or they must become annihilated in refractory tissue; or they must wheel in regular or irregular circuits which ultimately bring them back upon themselves. If all three of these things are constantly taking place, it seems plausible to picture the active areas of the brain as areas which are normally organized for straightforward transmission of impulses in well-defined pathways from afferent to efferent aspects of the tissue; whereas the silent areas of the brain may be defined as those tissues which are so organized that stimuli which enter them tend to circle through them in closed circuits. To some extent, and in certain parts, these tissue-types may be organized into macroscopically separate and circumscribed structures. In other regions, the tissues may be so intermingled that they can be recognized only under the microscope as cyto-architectural units.

To these three possible fates one may add one further possibility: that impulses which enter the silent system might engender there the accumulation of some sort of "nervous energy" or excitatory agent, at the same time undergoing extinction themselves. This accumulation of energy could then be looked upon as a potential source of involuntary movements, if its discharge were freed by disease from some normal pattern of regulation and "inhibition." (The word is placed in quotation marks because it implies an entirely new, undemonstrated, and hypothetical kind of "inhibition," which is little more than loosely analogous to the known manifestations of central or peripheral inhibition.) This speculation in some form might be said to be the secret assumption that runs through much current neurological thought. It it no less potent as a speculative assumption merely because it is left unformulated. Indeed, it would seem preferable to make a frank formulation of such an hypothesis, exploring its possibilities and its difficulties fully, instead of allowing it to colour our thinking in silence. The speculations presented in this paper are an alternative hypothesis, an effort to explore possible lines of investigation and explanation without recourse to the notion of the central nervous system as a storehouse of some unknown form of static "nervous energy," but rather as a site of multiple circuits of excitation waves, only some of which are normally in direct connection with the body as a whole.

If they exist at all, there must be countless such circuits; and at any one moment some will be stopping because they are encountering

refractory tissue, others starting, still others travelling at diminished intensity through tissue of partial refractivity, &c. And of all of this activity there may be outwardly no normal sign. It is, however, equally possible that the algebraic sum or the distribution of all of this activity may bear some relationship to many states of psychomotor activity—such as sleeping, waking, confusion, alertness, &c. It is attractive to speculate of such possibilities, but quite outside the main purpose of this paper.

On the other hand, it is part of its chief purpose to show how relatively easy it is to understand how such silent circus rhythms could act as a source of spontaneous involuntary movements. It is necessary to imagine only that the normal circuits through a silent area are altered by some pattern of lesions, or that the velocity of the wave or the duration of the refractory period is changed by some toxic or vascular or metabolic factor. As a result, normal pathways might be closed and unusual pathways opened which would lead the circling impulse out of its silent home into one of the active regions. What would happen then would depend upon the nature of the site which was invaded and the conditions met with there; but the passage of the wave through the active area would in each instance result in a discharge along some motor, sensory, or vegetative route. If the new circuit, once established, persisted unaltered—circling with a steady rhythm out of the silent area to touch the active area at a constant and limited point before swinging back again—the phenomenon of rhythmical clonic spasm could be readily understood (fig. 1). But if the circuits through the silent area were inconstant and tortuous, and if the points of escape were more than one, or if the active areas were played upon at more than one locus, the grotesque and irregular phenomena of chorea or athetosis would also seem to be explicable (fig. 2).

One may carry the speculation still further, and imagine that the escaping wave of excitation meets conditions in the active area which are so abnormal that instead of playing over the region merely for an instant it establishes here a temporary circus rhythm (fig. 3). If this occurs in the occipital cortex, is it hard to imagine that the result would be the scintillating phenomena of migraine, whose wheeling lights may be taken as giving support to some of these speculations? Or if certain cell layers of the motor system were involved, might not epilepsy be the expression of the process?

The duration of such a circus movement in an active area might

vary from a moment to hours or even days, depending upon the stability of the circus rhythm set up; which—in turn—would depend upon the fundamental variables of wave velocity, circuit length and duration of the refractory phase. An unstable, temporary circus rhythm would correspond to the ordinary isolated epileptic seizure. A more stable system might explain status epilepticus. And it may be that a closer approach to the therapeutic solution of these difficult problems will depend upon the finding of drugs which will alter in the central nervous

FIG. 1.

FIG. 2.

FIG. 3.

FIG. 1.—Circular excitation wave pursuing a regular and constant circuit, escaping from the silent system (shaded) to the active system (unshaded) always at the same point and at a constant rhythm. Such a pathological system could explain rhythmical clonic spasm.

FIG. 2.—Circuitous excitation wave pursuing an irregular and tortuous pathway, and escaping from the silent system (shaded) to the active system (unshaded) at multiple and varying points. Such a pathological system might account for athetosis or chorea.

FIG. 3.—Circular excitation wave escaping from the silent system (shaded) into the active system (unshaded) and setting up a temporary circus movement in the latter. Such an occurrence might underlie the epileptic attack.

system the relations of velocity to refractory phase, just as quinidine has been shown to act upon these factors in checking the probable circus movements of auricular fibrillation in heart muscle.

There would seem to be many implications of these speculations for pathology and for experimental physiology. If the speculations represent even an approximation of the true picture, it would no longer seem reasonable to seek for the pathological basis of such symptoms in a single lesion; rather would one expect that similar symptoms could arise from any of several patterns of multiple lesions, the effects of these lesions being influenced in turn by somatic factors which would alter

velocity, refractory periods, &c. A reconciliation between the neurogenic and somatogenic explanations of epilepsy might well evolve along these lines.

The immediate need, of course, is for experimental substantiation for these hypothetical closed circuits in the "silent" areas and a more intimate study of conduction of the excitation wave among the neurones of the brain. This presents obvious difficulties because here, as elsewhere, unless discharges proceed synchronously their electrical effects will tend to cancel each other. Such technical difficulties are not, however, insurmountable to modern methods.

It faces us, furthermore, with a fundamental problem as to the rôle which dendrites and neurones may play in conduction of the excitation wave. It is usual to think of the dendrites as the receiving antennæ of the neurone, with the axone as the only efferent channel, and of the nervous impulse as passing from the dendrites directly through the cell body, and out through the axone (see fig. 4). This may be true for the neurones of the active areas of the brain, and perhaps for all. But under abnormal conditions in the active areas, and perhaps even normally in the silent areas, it is possible to visualize the passage of an excitation wave in closed circuits from one neurone to another in one of two ways: either along one of the divisions of a branching axone (as in fig. 5), or along what would ordinarily be called their dendrites (as in fig. 6). In either case, as Sir Charles Sherrington suggests (personal communication), it is the Golgi Type II cell which would seem to lend itself most readily to this type of organization as a normal function. The second alternative, in which the circular excitation wave spreads from neurone to neurone along the dendrites, with effective discharges escaping from the circuit along the axones, involves a much more serious divergence from current assumptions; i.e., it implies that the excitation wave and axonal impulse may not be identical, that not all dendrites are exclusively receptive in function, that it may be possible for an axone to be in a refractory phase while its cell body and dendrites are active, &c. For most types of central neurones there exists no absolute evidence against such possibilities (except perhaps that which can be found in the recent paper of Denny-Brown and Cooper, for the irreversible conduction in the anterior horn cells). Nevertheless, it is probably well to hold this second possibility in reserve as the less likely alternative, depending rather upon the circuit-pattern which makes use of branching axones until such a pattern is shown to be insufficient.

It is possible to imagine that such circus movements might occur abnormally in many regions of the brain and cord, and even in

FIG. 4.

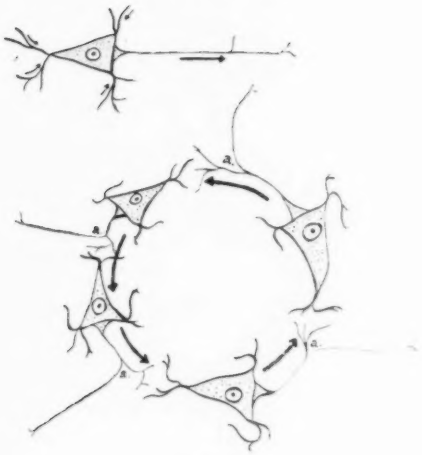

FIG. 5.

FIG. 6.

FIG. 4.—Accepted picture of conduction of the nervous impulse through the dendrites, cell body, and axone.

FIG. 5.—Diagram of an excitation wave circling through a group of neurones, and following the accepted ideas of conduction through polarized neurones and synapses, i.e., from axone to dendrites or cell body (as in fig. 4). Such a system would affect other regions of the CNS only through the impulses which escaped from the closed circuit along the branching axones (*a*).

FIG. 6.—Diagram of an excitation wave circling through a group of neurones according to a less orthodox pattern of conduction than in fig. 5. Here the wave passes from neurone to neurone along their dendrites, while the axones (*a*) lead out of the circuit and respond to the circling wave of excitation when they are not in a refractory phase. It is less likely that this is a normal form of circuit than that pictured in fig. 5.

sensory or vegetative ganglia. This leads one to wonder what rôle they might play in the production of spontaneous sensations, spontaneous pain, vegetative crises, &c. Nor is it difficult to imagine that they might take part in the production of such sustained disturbances as tremor, myoclonus, and fibrillation.

It is not inconceivable that among certain neurones they may support normal functions. The relatively autonomous rhythm of discharge along post-ganglionic fibres of the sympathetic system is understandable on such a basis. The tetanic nature of the crossed extension response to a single afferent stimulus is not difficult to explain by means of a circus rhythm which repeatedly fires the efferent cells. Even in the phenomena of tonus and tonic perseveration there is room for these speculations. One is tempted, furthermore, to point out that in the production of these phenomena it would make a great difference whether the circling waves were synchronous or asynchronous, rhythmical or arrhythmical (i.e., constant or varying orbits), confined to a group of synergic neurones or traversing the boundaries between antagonistic groups. But perhaps it is better to resist the impulse to exploit the idea any further until some fragments of experimental support should have accumulated.

In conclusion, these speculations may be reduced to a series of propositions.

(1) The brain consists of two types of tissue which can be called respectively the "active" and the "silent" systems of the brain.

(2) To some extent, and in certain parts, these tissue-types may be organized into macroscopically separate and circumscribed structures. In other regions, the tissues may be so intermingled that they can be recognized only under the microscope as cyto-architectural units.

(3) By "active" tissue is meant all of those neurone systems which are so intimately and directly related to the rest of the body, that the passage of an impulse along any of them always registers in some way in the body economy. There can be no play of impulses along the neurones of this active system which is not directly referable to the exterior of the central nervous system, either along afferent or efferent pathways.

(4) The neurones of the active system may be organized, under normal conditions, strictly according to the neurone doctrine, i.e., with dendrites afferent to the cell body and axone efferent from it, and with irreversible synapses. (The possibility must be borne in mind that under abnormal conditions this organization for through-and-through

conduction with irreversible synapses may be altered, and that there may be some departure from this, even normally.)

(5) Under normal conditions impulses impinge upon the active system only or chiefly from stimuli arising from without the central nervous system.

(6) Under abnormal conditions impulses may arise within the active system from stimuli which reach it from the silent system of the brain, causing spontaneous movement and sensation, or spontaneous vegetative alterations.

(7) This necessitates speculation as to what possible sources may exist in the silent system for such an aberrant stimulus :—

(*a*) The silent tissues of the brain are defined as those through which impulses may pass without any external registration of their passage.

(*b*) Until extinguished by meeting with refractory tissue, such impulses will continue to circulate indefinitely.

(*c*) This implies the existence of circuitous pathways through the silent system—pathways which may be long or short, regular or tortuous, constant or varying—but which must ultimately return upon themselves.

(*d*) Whether the impulses which travel along the pathways of the silent system are produced by some hypothetical form of spontaneous generation of impulses within this system, or whether they result from a diversion of proprioceptive impulses out of the active afferent system into the silent circuits, is immaterial to the main speculation, which is that there is a silent system in which such circling impulses normally exist.

(*e*) Pathological states which divert these silent circling impulses out of their normal silent pathways into the active system offer a plausible explanation of spontaneous sensation or movement, whether the latter be chorea, athetosis, rhythmical clonic spasm, or epilepsy. In the latter, the speculation surmises the establishing of an irregular, transient circus movement in the active motor system.

(*f*) Such pathological states may be a combination of local lesions, or merely a chemical change which alters the relations of conduction velocity, duration of refractory phase, intensity, and the length of the circuit.

(8) These speculations suggest many departures from the tacit assumptions of current neurological thought—departures which imply a somewhat less rigid adherence to the neurone doctrine than is customary,

without, however, demanding that this fruitful doctrine be abandoned *in toto* :—

(*a*) Not all neurones must necessarily be organized for through-and-through conduction, with irreversible synapses. Those of the silent system may be organized for more circuitous and diffuse conduction systems.

(*b*) Even within the active system it is conceivable that conduction in closed circuits may play some normal rôle—and certainly it must be reckoned with as one of the possible explanations of abnormal functions. (This paper has attempted to give illustrations of possible examples of such disturbances.)

(*c*) The excitation wave in dendrite and neurone may not be identical with the axonal impulse, as is usually assumed. The laws of conduction within the neurones of the central nervous system must, therefore, be studied as intensively as has been done in the peripheral axone.

(*d*) The circling excitation wave may be closely related to the excitatory agent which Sherrington has found it necessary to postulate, and inhibition may find its mechanism, as Keith Lucas suggested, in some expression of the refractory phase ; to which the suggestion is added here that it may be of the refractory phase, or some other fundamental attribute, of the circling excitation wave.

In reviewing the development of these ideas in my own mind, I find one special indebtedness which I wish to acknowledge. I had for some years been ruminating about what rôle circus movements might play in the central nervous system—thinking especially about such rhythms among the cells of the brain-stem, cord and basal ganglia. In the autumn of 1929 my friend, Dr. Jonas Friedenwald (Ophthalmic Surgeon and Pathologist to the Wilmer Ophthalmological Institute of the Johns Hopkins Hospital) made the observation to me that the wheeling lights of his own migrainous headaches made him think of circus movements in the occipital cortex—and that by analogy epilepsy might be explicable in a similar way. This stimulated me to the present effort ; and for the rest of these speculations, as presented here, I must take the blame ; except that I acknowledge with gratitude the critical objections and suggestions of Drs. John Fulton, David Rioch, E. G. T. Liddell, and D. Denny-Brown, all of which I have tried to reply to or incorporate here. Finally, it is a pleasure to express my gratitude to Sir Charles Sherrington for his fundamental criticisms and for the encouragement without which I would have hesitated to publish these speculations.

THE INFLUENCE OF EFFERENT CEREBRAL PATHWAYS UPON THE SYMPATHETIC NERVOUS SYSTEM.

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Introduction.

Study of the anatomy and physiology of the autonomic nervous system has always been difficult. From the morphological point of view the peripheral nerves and ganglia are diffuse and do not lend themselves, as a whole, to satisfactory macroscopic or microscopic examination. Knowledge of the physiology of the autonomic system has been illuminated by the discovery that certain drugs have specific action upon its activity.

Certain data concerning the general plan of the peripheral autonomic reflex arc are now available. Small motor cells found in certain parts of the brain-stem and cord give rise to the preganglionic neurones which connect in the periphery of the body with the postganglionic neurones. Afferent impulses are carried from the structures innervated by the autonomic fibres to the central nervous system and are supposed to influence the reflex activity of the motor cells.

There is much less information concerning the control of this primary reflex arc by the higher correlation centres of the brain. It has become clear, however, that abnormalities of the structures in the floor of the third ventricle, following invasion by tumours or injury in lethargic encephalitis, for example, cause metabolic disturbances influencing weight, sleep, fluid intake and output, vasomotor control and sweating. The conclusion, therefore, seemed justified that groups of cells in this area form a general central controlling centre for autonomic activities.

The pathways that carry the impulses from centres in the hypothalamus to the preganglionic autonomic cells of the brain-stem and cord have never been clearly demonstrated. These pathways are non-

medullated and are believed to consist of chains of neurones with numerous synaptic junctions.

The present paper attempts to determine some of the influences from the brain that control the primary autonomic reflex arcs. The experiments recorded here have to do only with the phenomenon of sweating. Therefore specific attempts to study the cerebral control of the autonomic system will be recorded only inasmuch as they have to do with sweating. Moreover, stimulation of the sweat-glands is a function of only one group of cells of the autonomic system, the sympathetic cells of Langley's classification.

A.—Previous Studies of the Cerebral Control of Sweating.

Bechterew (1905 and 1911) found that faradic stimulation of certain areas in the anterior portion of the cerebral cortex, close to the motor area, in cats produced sweating of the foot-pads which could be demonstrated on inspection and with the use of litmus paper. The secretion occurred in all four extremities, although it was more marked in the extremities of the opposite side. A diagram (1911) shows the exact position of the two circumscribed cerebral areas from which this phenomenon was obtained. If these areas be projected upon the drawing of the cerebral cortex of the cat in the present paper (fig. 1), one corresponds to areas A and G and the other to area D. Bechterew pointed out that sweating was a frequent accompaniment of mental activities, particularly under certain pathological circumstances. He also collected cases in which sweating of the opposite side of the body was associated in man with glial tumours infiltrating the frontal lobe and encroaching on the motor cortex.

Winkler (1908), working with the cat, confirmed these observations of Bechterew. He, moreover, exposed the floor of the fourth ventricle and obtained profuse sweating of the foot-pads of all four feet on faradic stimulation of an area one millimetre cephalad to the calamus scriptorius. Faradic stimulation of the cerebral peduncles also elicited sweating of the foot-pads.

Karplus and Kreidle (1909) demonstrated that stimulation of the tuber cinereum, behind the optic tract and lateral to the infundibulum, elicited, in the cat, dilation of the pupils, retraction of the nictitating membranes, secretion of tears, a rise in the general arterial blood-pressure and profuse sweating of the pads of all the feet. They assumed, without any adequate reasons, that they stimulated the

subthalamic nucleus and thought this nucleus was of autonomic function.

In all of these experiments the effect on sweating produced by faradic stimulation of the cortex or brain-stem was judged either by the presence of visible sweat or by changes in the litmus paper applied to the skin. These criteria have obvious limitations. In the first place a fairly strong and continuous stimulation is necessary to produce visible sweat, and then the sweat must be wiped off before each repetition of the stimulation. This method does not permit of an actual measurement of the effects produced by the stimulus, nor of photographic registration of the types of response.

B.—A Delicate Method of Recording the Activity of the Sweat-glands on the Foot-pads of Cats.

Through the galvanic skin response method of studying the activity of the sweat-glands most of these limitations are overcome. By this method the activity of the sweat-glands is measured indirectly through the action currents given off from the glands when they are activated. The close correspondence between activity of the sweat-glands and the galvanic skin response has recently been clearly demonstrated by Darrow (1927). The extraordinary degree of sensitivity of this method can be seen by the fact that even a slight emotional or nocuous stimulation in an intact animal or in man is sufficient to cause a galvanic current to be given off by the sweat-glands. Also, a small make and break shock applied to the sympathetic chain or to the base of the brain produces a definite galvanic skin response (Wang and Richter, 1928).

The technique of obtaining this reflex has been described in detail in previous papers (Richter, 1927, 1928, 1929 and 1930). Only a brief description of the essentials will be given here. The electrodes, zinc discs and kaolin paste, are attached to the skin on the large central pad of the foot and to the pinna of one ear, the skin of which had previously been pierced with a hypodermic needle. In this way responses from the ear are eliminated and only the responses from the pad of the foot are obtained.

The electrodes on the pad of the foot and the ear are connected to a string galvanometer of the same type used in cardiac departments. No external current is employed, so the currents that are given off have their origin in the sweat-gland activity in the skin of the pad of the foot. Two records of the galvanic skin response produced by stimula-

tion of the brain-stem and cortex are shown in fig. 15. Time in seconds is indicated at the top of the records, the incidence of the stimulus (faradic current) at the bottom, and the galvanic current in the middle. It may be noted that the latency of the responses is about one second, which clearly distinguishes them from muscular responses. These records will be discussed again below.

C.—The Location of the Motor Cerebral Cortex of the Cat.

In recent experiments (Weed and Langworthy, 1926, and Langworthy, 1927) attempts have been made to map out the motor cerebral cortex of the cat by the method of faradic stimulation, and also to

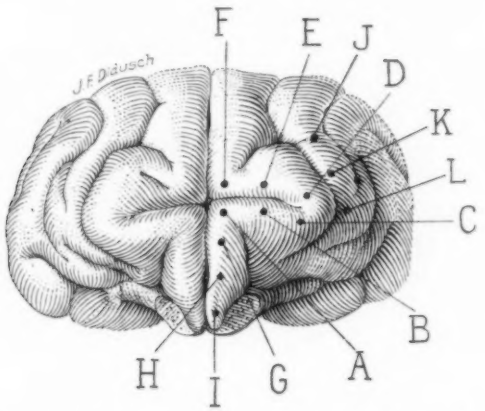


Fig. 1.—Frontal view of the cerebral cortex of the cat. Galvanic skin responses were obtained on faradic stimulation of areas A and G and areas D and K. Contraction of the contralateral somatic musculature was elicited from areas A, B, C, E and F.

follow the development of responses from the motor cortex in young kittens. The results may be interpreted by consulting fig. 1. Stimulation of areas A and B produced flexion of the contralateral fore-leg, of areas E and F flexion of the contralateral hind-leg, while stimulation of area C elicited contraction of the facial and masticatory muscles of the opposite side. No motor response was ever obtained on stimulation of area D.

Afterwards it was shown that removal of the area frontalis, including areas A, G, H, and I of fig. 1, produced abnormalities in tonus of the contralateral legs, manifest as hypertonus (King, 1927, and Langworthy, 1928). It was suggested that the fronto-ponto-cerebellar tract

arises from cells in this area and abnormalities in tonus occur as a release phenomenon after its injury.

It was possible to use this previous work as a basis for studying areas in the cerebral cortex from which the action currents could be demonstrated after faradic stimulation, and to correlate the location of these new areas with those to which attention had already been directed.

Methods.

An attempt was made in this study to survey the cerebral cortex and brain-stem for areas from which activity of the sweat-glands could be elicited by Faradic stimulation. Thirty-six cats were used in these experiments. The cat was given ether at the beginning of the experiment and remained under the anæsthetic until after decerebration. Inasmuch as decerebrated cats no longer are conscious or feel pain, the ether was discontinued. Artificial respiration by a tracheal tube was used when sections of the lower brain-stem were made. In many cases the carotid arteries were tied to limit the bleeding; pressure on the vertebrals also helped to control the hæmorrhage.

At the beginning of the experiment the cerebral cortex was exposed on each side. Stimulation of the cortex was not confined to the region of the motor area, but the whole dorso-lateral surface of both hemispheres was explored. After the results from the intact cortex had been ascertained, transections of the brain were made with a sharp knife. These sections were not exactly coronal but sloped in an anterior direction towards the ventral surface. After bleeding had been controlled, the whole cut surface was carefully explored with the stigmatic electrode. In each animal three or four of these transverse sections were studied successively before the tentorium was reached. At this point the tentorium was cut away and the bone removed from the whole dorsal surface of the cerebellum. After faradic exploration of the dorsal surface of the cerebellum, the cerebellum was removed entire by severing the peduncles. This exposed the whole floor of the fourth ventricle which was explored for areas from which galvanic skin responses could be obtained. Areas which gave a response are shown in fig. 2 (areas 1 and 2).

The lower portion of the brain-stem was now exposed, permitting further transverse sections. Artificial respiration gave proper oxidation of the blood and the animals remained reflexly responsive. It was possible to continue the study of transverse sections into the region of the cervical cord.

A faradic current from an induction coil was used for stimulation of the central nervous system. A large indifferent electrode was attached to the back of the animal. The stigmatic, stimulation electrode consisted of a fine platinum point mounted on a delicate spring. It could be demonstrated that relatively little spread of the current occurred around the point of stimulation. The strength of current

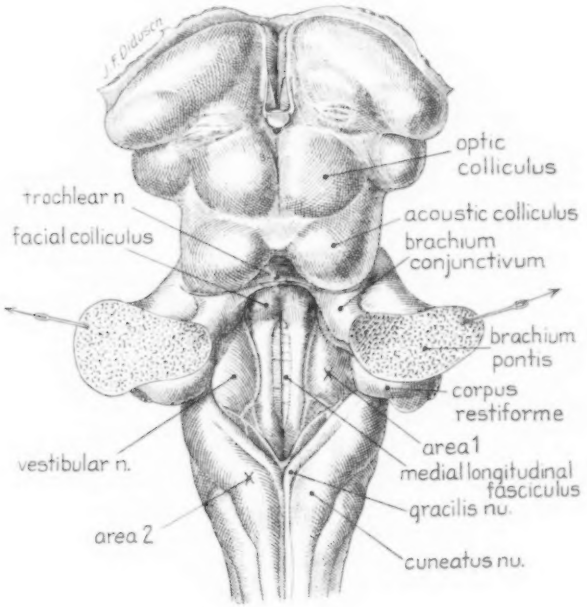


FIG. 2.—Drawing of the floor of the fourth ventricle of the cat. Galvanic skin responses were obtained from the region of the vestibular nuclei (area 1) and from the posterior column nuclei (area 2).

necessary to obtain the reflex was approximately the same as that required to elicit a motor response. In several instances it was much smaller.

EXPERIMENTAL OBSERVATIONS.

A general survey of the central nervous system was made for groups of cells or tracts of fibres from which responses could be elicited on faradic stimulation. The results may be described under a number of headings.

(*a*) *Stimulation of the Cerebral Cortex.*

The whole dorso-lateral surface of the cerebral cortices was explored with the stigmatic electrode for areas from which stimulation of the sweat-glands could be obtained on stimulation. The two areas that gave autonomic responses were sharply circumscribed and were adjacent to the motor cortex from which contraction of the contralateral musculature may be obtained. They corresponded almost exactly to the areas previously described by Bechterew. One of these areas was anterio-medial to the motor cortex, occupying areas A and G of fig. 1. The maximum response was obtained in the boundary between areas A and G. The second area was just posterio-lateral to the motor area, which gives responses of the contralateral striated musculature. It occupied areas D and K of the diagram (fig. 1), with the maximal response from area K. No action current was obtained on stimulation of any other portions of the cortex.

As has been described, the responses were recorded quantitatively in terms of deflection of the galvanometer string. Approximately equal responses were obtained from these two areas. On stimulation of one of these points in one hemisphere, action currents could be obtained from all four legs, although they were more marked on the contralateral side. The fore-legs gave more marked responses than the hind-legs.

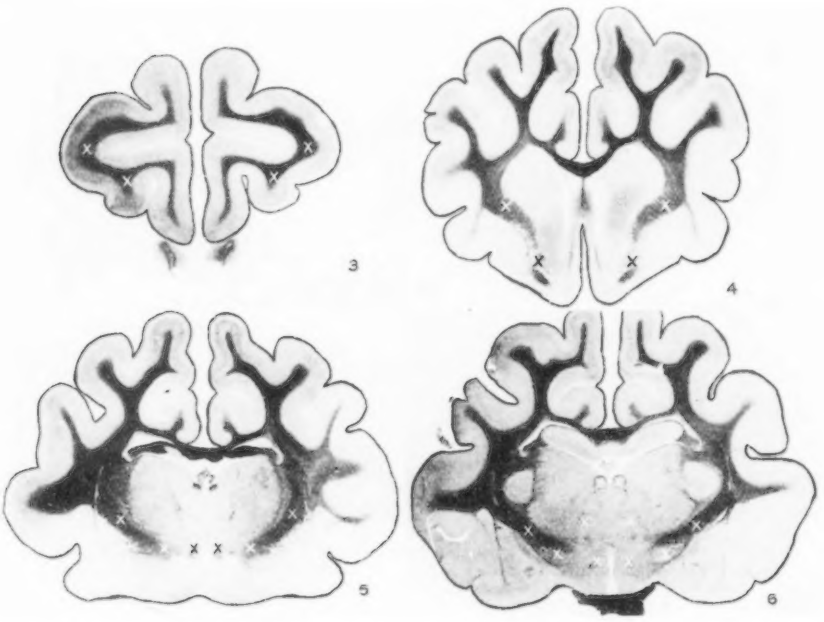
Before attempting to follow tracts from these areas from experimental level to level of the brain-stem, it is important to seek some interpretration of the possible connections of these areas. As already suggested, there is some evidence that areas A and G give rise to the cortico-pontine fibres, influencing the tone of the striated musculature through the cerebellum. This pathway lies mesial to the cortico-spinal tract in the internal capsule. A temporo-pontine tract descends just lateral to the cortico-spinal fibres to connect with the cerebellum in a similar way. It may be said that areas D and K give rise to the temporo-pontine fibres.

(*b*) *Faradic Stimulation at Different Experimental Levels of the Fore-brain.*

The areas from which galvanic skin responses were elicited from transverse sections of the fore-brain are shown in figs. 3 to 7. The first section (fig. 3) is just caudal to the motor area. Two circumscribed points were constantly localized in each hemisphere; responses could

be obtained from each of them. Their relation to the cortico-spinal tract becomes clear if they are followed in figs. 4, 5 and 6. It is evident that one group of fibres lies mesial, the other lateral to the cortico-spinal tract. This again suggests that they are fronto- and temporo-pontine tracts. They may be followed into the region of the mid-brain where all three cortico-efferent tracts become extremely compact (fig. 8) and can no longer be differentiated by faradic excitation.

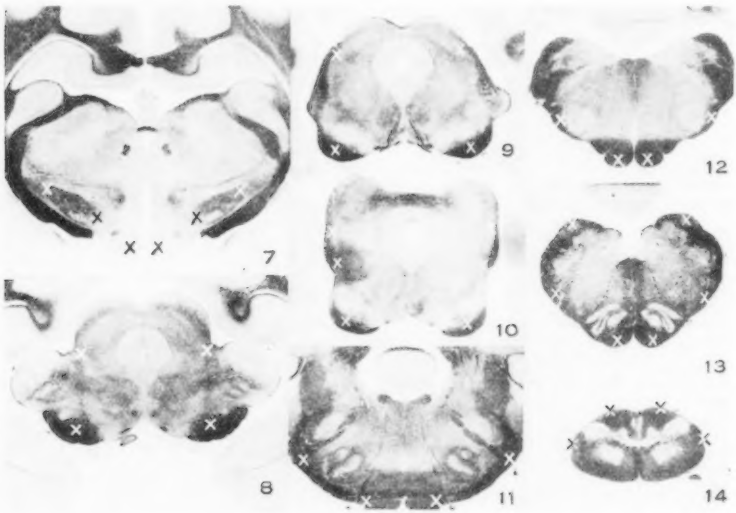
At the levels of the fore-brain corresponding to figs. 3 and 4, the



only responses were from areas that corresponded with tracts from the cortical areas already described. Other responsive points appear in figs. 5, 6, and 7. A group of these points lies near the ventral mid-line, around and in the floor of the third ventricle. In fig. 5 the third ventricle is a narrow median slit; this section is anterior to the optic chiasma which is seen in fig. 6. Just lateral to the third ventricle and ventral to it galvanic skin responses were obtained on stimulation. The area just ventral to the optic chiasma was responsive in fig. 6; also an area in the mesial portion of the thalamus and the region of the

habenular nuclei at the dorso-medial edge of the thalamus. In fig. 7 a responsive area in the tuber cinereum is charted.

The ventral or hypothalamic area from which responses were obtained includes the whole tuber cinereum region. This area lies just between the cerebral peduncles, and the question may be raised as to whether the effects obtained were not due to spread of current into the tracts that have been followed from the cerebral hemispheres. Against this view is the fact that large deflections of the string were elicited from the tuber cinereum, greater than from the region of the



FIGS. 3 to 14 are photographs of coronal sections of the brain of the cat stained by the Weigert-Pal method. The areas which gave galvanic skin responses on faradic stimulation are indicated. A discussion of these areas will be found in the text.

cerebral peduncles. There are, then, groups of cells in the tuber cinereum which, on stimulation, produce sweating of the foot-pads.

Nothing more need be said here concerning the areas in the thalamus and habenular nuclei from which action currents were obtained. These will be discussed later.

(c) *Galvanic Skin Responses obtained on Stimulation of Transverse Sections of the Mid-brain.*

Three figures demonstrate typical transverse sections of this region; fig. 8 from the extreme cephalic end of the mid-brain, fig. 9 through

the optic colliculi, and fig. 10 through the acoustic colliculi. In each case stimulation of the cerebral peduncles on the ventral surface of the sections gave a marked response. Due to the condensation of fibres in this area, the two separate pathways from the cerebral cortex can no longer be differentiated.

In figs. 8 and 9 it will be observed that a galvanic skin response was always obtained from an area on the lateral edge of the brain-stem just ventral to the optic colliculi. This pathway may be followed into fig. 10, where it forms the most dorsal area from which responses were obtained. It could not be followed further caudad in the brain-stem. The connections of this tract are not clear.

Fig. 9 is at the level of the red nucleus. Stimulation of the red nucleus never elicited action currents. The rubro-spinal tracts below this level, however, always gave a marked response on stimulation. This response was more marked than that obtained from the cortico-spinal fibres in the medulla. Thus the lateral area in fig. 10, between the dorsal and ventral points already discussed in the two preceding paragraphs, gave a marked response. This same area can be traced caudad in the other sections (figs. 12 to 14).

(d) Galvanic Skin Responses obtained on Stimulation of the Floor of the Fourth Ventricle.

After the cat was decerebrated by a transverse section through the mid-brain, the dorsal surface of the cerebellum was exposed. No response was ever obtained on stimulation of the cortex of the vermis or hemispheres of the cerebellum. The cerebellum was then removed in one piece by cutting the peduncles and the floor of the fourth ventricle exposed to view.

Stimulation of the floor of the fourth ventricle and its environs gave marked galvanic skin responses, and confirmed and amplified the findings previously made by Winkler. The responsive areas are indicated in fig. 2 (areas 1 and 2). No response was obtained from the cephalic portion of the floor of the ventricle, from the region of the facial colliculi, caudal end of the aqueduct, or from the dorsal surface of the acoustic and optic colliculi. From an area just medial and caudal to the cut cerebellar peduncles responses were obtained on faradic stimulation (area 1). These bilateral responsive areas include lateral prominences at the widest point of the fourth ventricle. They are the vestibular nuclei which give rise to the vestibulo-spinal tracts and certain fibres running caudal in the medial longitudinal fasciculus.

The most marked action currents obtained anywhere were elicited from the region of the nuclei gracilis and cuneatus just lateral to the caudal portion of the fourth ventricle (area 2). In certain of the cats, relatively unresponsive to stimulation of other portions of the brain, galvanic skin responses were always obtained from these areas. This marked response was obtained, not only from the posterior column nuclei, but also from the surface of the posterior columns throughout the cervical cord.

Between the vestibular nuclei and the posterior column nuclei there was always an area unresponsive to stimulation. No responses were obtained from the medial portion of the floor of the fourth ventricle.

(e) *Galvanic Skin Responses obtained on Faradic Stimulation of Transverse Sections of the Medulla.*

Three typical sections of the medulla are shown in figs. 11, 12 and 13. The ventral areas in each section from which responses were obtained correspond to the location of the cortico-spinal tracts. More marked responses were always obtained from a lateral area marked in each section which corresponds to the region of the rubro-spinal tract.

A third area is marked in fig. 13; maximal responses were always obtained from this region of the posterior column nuclei. This has already been discussed in connection with the surface study of the region of the fourth ventricle floor.

(f) *Galvanic Skin Responses obtained on Faradic Stimulation of Transverse Sections of the Upper Cervical Cord.*

Marked responses were obtained on faradic stimulation of transverse sections of the upper cervical cord. These may be described in connection with a section at the highest cervical level (fig. 14). In the cat the rubro-spinal and cortico-spinal fibres all run in the lateral columns; they are more or less intermingled there. Galvanic skin reflexes were obtained from faradic stimulation of the lateral columns in the region of the cortico-spinal and rubro-spinal pathways. The most marked responses, however, were always obtained from the posterior columns. No deflection of the galvanometer string was ever obtained from the ventral columns.

Discussion.

Certain conclusions concerning cerebral influences upon the preganglionic sympathetic cells of the cord may be drawn from these

experiments. It is evident that stimulation of the cortico-spinal, cortico-pontine, rubro-spinal and vestibulo-spinal tracts elicit action currents indicating stimulation of the sweat-glands. It appears, therefore, that the efferent pathways from the brain influence the activity of the autonomic cells in the anterior grey matter of the cord.

This is not a strange, nor should it be an unexpected finding. For many years it has been known that many efferent pathways from the brain influence the somatic motor anterior horn cells, and modern physiological studies give certain clear ideas concerning their functions. Efferent pathways to the cord from the medulla control the tonic patterns of the striated musculature of the extremities in reference to stimulation of the vestibular and optic reflex centres. The cortico-spinal pathway initiates delicate phasic movements, while at the same time there is reason to believe that the cortico-pontine influences inhibit the tonus of the antagonistic group of muscles to make these movements possible. The sympathetic cells lie close to the somatic motor cells in the anterior columns of the cord, so it is not surprising to find that they should be activated by the same cerebral influences. This seems the more comprehensible inasmuch as no cerebro-spinal pathways ending exclusively around the sympathetic cells are known.

The influences of cerebro-spinal pathways upon the somatic motor cells of the cord are probably summated to produce either excitation or inhibition of the activity of the anterior horn cells, which in turn control the tonus and contraction of the somatic musculature of the body. It is important to learn whether evidence of excitation and inhibition can be obtained by recording the action currents in the case of the sympathetic cells of the cord.

Galvanic skin responses similar to those described in this paper may be obtained from normal men and from intact animals with the proper stimulus. For example, firing a pistol close to the human subject will elicit such a response. In man, a response is obtained by reading a list of words, some of them giving strong emotional reactions. The galvanic skin reflexes obtained under these conditions are usually biphasic, consisting of both a positive and negative phase. In the present experiments biphasic currents were obtained in almost all of the animals on stimulation of the cerebral cortex; responses from other areas in the brain were uniphasic, of only a negative phase. Records are shown in fig. 15 of the biphasic responses obtained from cortical stimulation and the monophasic responses from subcortical stimulation. This question of positive and negative responses will be studied further.

There is good evidence, however, that the cerebral control of the autonomic system is predominantly an inhibiting one. Riddoch (1917) and Head and Riddoch (1917) had a unique opportunity to study selected cases of complete transverse section of the spinal cord in young

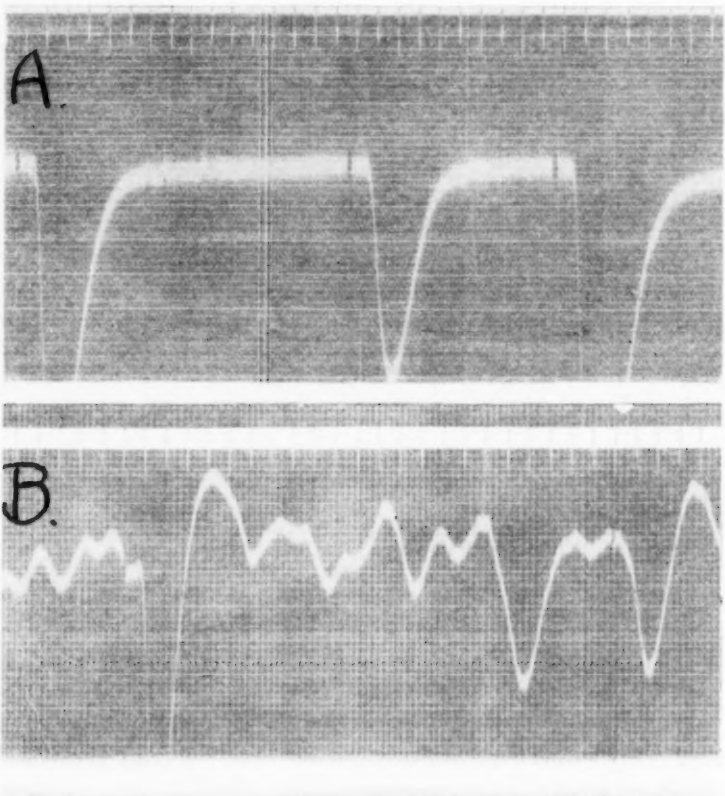


FIG. 15.—Typical galvanic skin responses obtained on faradic stimulation of the cerebrum of the cat. A monophasic response (*a*) and a biphasic response (*b*) are shown. Biphasic responses were obtained on appropriate stimulation of the cerebral cortex. After removal of the responsive areas in the cerebral cortex, monophasic responses were obtained.

human adults, otherwise healthy, in whom the injuries were produced by war wounds which healed rapidly with little infection

For several days following the injury the musculature innervated by the cord below the level of the lesion was completely atonic and no reflexes could be obtained. Similarly, the smooth muscle of the bladder

lost its tone. The skin at this time was very dry and no sweating occurred. After a variable period of seven days or more after the injury, reflexes began to return in the lower portion of the body, and later the reflexes presided over by the cord below the lesion became hyperactive. At this time stimulation of the skin produced not only a local contraction of the musculature, but violent mass contraction of all the striated musculature, evacuation of the bladder and sweating over the portion of the body innervated below the lesion. Riddoch pointed out that the reflex arcs lost their local sign and a mass reflex resulted. The profuse sweating indicated that the sympathetic cells of the cord, freed from normal cerebral inhibitory control, became hyperactive.

Richter (1930) has recently demonstrated in animals that the skin resistance is greatly increased and the galvanic skin reflex is absent after transection of the cord in the upper thoracic region for the first few days following the operation. This is the period of spinal shock described by Riddoch in man. Later, when reflexes return and become hyperactive in the posterior portion of the body, the skin resistance drops to normal, or below normal, and the galvanic skin reflex becomes hyperactive, indicating the marked activity of the sweat-glands.

It is clear, then, that following transverse section of the cord, after the period of shock has passed, reflex activity of the autonomic, as well as of the somatic, motor cells becomes greater than normal. This suggests that the cerebral control of autonomic activity in the cord is predominantly an inhibitory one.

In the present experiments responses were obtained from areas in the floor of the third ventricle, from the habenular nucleus and from a mesial area in the thalamus. It is impossible to suggest the anatomical pathways that would carry impulses from these regions to the motor cells in the cord. The reflex path probably connects with cerebro-efferent pathways lower in the brain-stem, the rubro-spinal tract for example.

The most marked galvanic skin reactions were obtained on stimulation of the posterior column nuclei and the posterior columns of the cord. These fibres must make important reflex connections with the sympathetic cells of the thoracic cord.

It seems likely that the two areas in the cerebral cortex from which galvanic skin responses were obtained corresponded to the regions giving rise to the fronto- and temporo-pontine tracts. One area, the frontal, lies just antero-mesial to the true motor cortex; the temporal, postero-lateral to it. There has been previous evidence that the anterior

area gives rise to the fronto-pontine tract (Langworthy, 1928). Although these pathways connect with the cerebellum, no action currents were obtained on stimulation of the cerebellar cortex. Stimulation of the related rubro-spinal tracts gave marked reactions.

The galvanic skin responses obtained were always larger from the fore-legs than from the hind-legs. Although the responses were always bilateral, they were usually unequal on the two sides. Those from the cortico-spinal tract were predominantly bilateral, from the rubro-spinal tract and floor of the third ventricle predominantly unilateral.

The responsiveness of the cats varied considerably. A few gave no response at all; others gave only small responses. The impression was gained that the reflex was practically abolished in a period of very deep anæsthesia. The sensitivity of the response could be heightened by keeping the animals in a hot-air bath for several hours before the experiment.

Finally, the conclusion seems justified that the same pathways from the cerebrum that influence and control the somatic motor primary reflex arcs also influence and control the primary sympathetic reflex arcs. Although there may be higher centres for sympathetic control in the brain, these finally exert their influences through the pathways described in this paper. It is clear from observations upon man and animals after complete transection of the cord that this influence must be predominantly an inhibitory one.

Summary.

Using the galvanic skin response as a measure of sweat-gland activity, the controlling influences from the cortex, brain-stem and spinal cord were investigated. Definite galvanic skin responses were obtained from faradic stimulation of two areas adjacent to the motor cortex, from the floor of the third ventricle, from the cortico-spinal and rubro-spinal tracts, from the vestibular nuclei, from the posterior column nuclei and from the posterior columns of the cervical cord.

It is concluded that the same cerebro-efferent pathways which influence somatic motor cells also control preganglionic sympathetic cells; this cerebral control is predominantly an inhibitory one. Any cerebral centres influencing lower centres seem to exert final control through well-known cerebro-efferent pathways.

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A CLINICAL STUDY OF TUMOURS INVOLVING THE OCCIPITAL LOBE.

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(From the National Hospital, Queen Square, London.)

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I.—Introduction.

Contributions to the literature of tumours of the occipital lobe have been very few, especially during the last ten years. They have been confined to an occasional case report and to two extensive papers—the first by Puusepp [22], not available to the majority of readers, and the second by Frazier and Waggoner [9], as yet available only in abstract. The former paper was based on a series of eight cases, and the latter on a series of forty cases, in nine of which the tumour had originated in the temporal lobe or had invaded the occipital lobe from its origin in the parietal lobe.

It has been thought necessary, therefore, to review the clinical features of a series of tumours of the occipital lobe, and to consider particularly certain points connected with the variations in the visual fields.

This paper is based on material from the National Hospital, Queen Square, London. The writer wishes to take this opportunity of expressing his thanks to the Medical Committee of the Hospital for permission to make use of the material and to members of the Staff for permission to quote cases under their care.

The limitations to be placed upon the choice of cases for study demanded some consideration, and eventually it was decided to base the review on a series of forty cases, the sole condition on which the cases were to be included in the series being that the tumour should have been proved anatomically to have involved the occipital lobe.

A further point for consideration was whether the series should be confined to those cases in which the occipital lobe alone was involved during the whole of the clinical course. A preliminary survey of the material showed that this course was undesirable if a complete account of the clinical features of occipital tumours was to be given. The importance of neighbourhood symptoms and signs was at once evident, so it was found necessary to include in the series for study those cases of cerebral tumour which involved the occipital lobe either primarily or chiefly, whether confined to the occipital lobe or involving other structures as well.

Of the series of forty cases of tumour of the occipital lobe studied, in twenty-four the tumour was entirely confined to that lobe, in six it involved the posterior part of the temporal lobe as well, while in ten it apparently affected a portion of the parietal lobe in addition to the occipital lobe.

It was felt to be inadvisable, on the limited clinical material available, to classify the clinical features according to the position of the tumour in the occipital lobe, as thereby a false impression might be created as to the relative importance of the different clinical pictures. This aspect of the subject will, however, be considered in the discussion of the individual symptoms and in the general discussion of the clinical features later in this paper.

Tumours involving the occipital lobe are relatively uncommon. Dowman and Smith [7] mentioned only two cases in a series of 100 verified intracranial tumours reviewed by them. The incidence of such tumours at the National Hospital during the past ten years has been practically the same or perhaps a little lower. In his review of a year's work at Cushing's Clinic, Cairns [3] mentioned nine cases as having been found in the group of 162 intracranial tumours verified either at operation or at autopsy.

In spite of their comparative infrequency tumours of the occipital lobe are of considerable importance clinically, not only because of the need to localize and deal with them satisfactorily, but also in the differential diagnosis of parietal, temporal, suprachiasmal, cerebello-pontine angle and cerebellar tumours, and of vascular conditions involving the occipital lobe.

The nature of the tumours in the series studied was as follows: In sixteen cases some form of glioma was present, in one an angioma, in three a sarcoma, in two each a gumma and a cyst, and in four an

endothelioma. In the remaining cases the nature of the tumour was not recorded.

II.—Review of the Literature.

The first reference in the modern literature to the clinical features associated with a lesion of the occipital lobe other than those due to a vascular condition was that of Levick [18] in 1866, who described a patient with left homonymous hemianopia due to an abscess about 1½ in. in diameter on the convex surface of the right occipital lobe. Subsequently, Jastrowitz [16] reported a case of sarcoma of the left occipital lobe involving all three of the convolutions and the precuneus, and associated with a wedge-shaped area of softening passing through the white substance to the posterior cornu of the lateral ventricle. In addition to vertigo, loss of memory and partial agraphia with transient right hemiplegia, the patient had right homonymous hemianopia with preservation of central vision in both eyes. At the same time Jany [14] reported a case of cystic glioma in the left occipital lobe, which had destroyed the cortex of the lobe at its apex and also the entire inner half of the lobe. This patient also had right homonymous hemianopia with preservation of central vision. Traube [28] described a cyst the size of an apple in the white matter of the right occipital lobe in a patient who had had epileptic attacks for seven years, with later an aura of darkness coming over the visual field from the left side. Haab [10] also described two cases. The first was that of a tumour of the inner surface of the apex of the right occipital lobe. There was a zone of yellow softening limited to the cortex. The patient had been irritable and stupid, for two months had had general convulsions, and on examination was found to have left homonymous hemianopia. In the second patient there was a cyst surrounded by an area of softening, 6 cm. by 3 cm., on the inner surface of the apex of the right occipital lobe. There was left homonymous hemianopia, the field of vision in both eyes being limited by a vertical line passing through the fixation point.

Hale-White [11] surveyed the pathological material of Guy's Hospital for the years 1872-1884 inclusive, and found an incidence of one cerebral tumour to every 59·1 cases coming to autopsy. Among the 100 cases of cerebral tumour discussed there were six in which the tumour involved the occipital lobe. Of these two were secondary to tumours elsewhere in the body and two were associated with tumours in other parts of the brain. Insufficient clinical data were recorded for these cases to be of any value in discussion.

Starr [25] discussed the visual area of the brain as determined by

a study of hemianopia, and quoted from the literature a series of cases of occipital tumour, including those of Jastrowitz, which involved the cortex of the calcarine fissure either alone or with destruction of other parts of the occipital lobe.

Harris [12] discussed hemianopia with special reference to its transient varieties and considered hallucinations, both of the formed and unformed type, occurring in the hemianopic fields of vision.

In a discussion on the localisation of intracranial tumours, Bramwell recorded the details of four cases of tumour of the occipital lobes, all of them of considerable interest in connection with the more unusual manifestations of tumours in that region. In one case the tumour was confined to the occipital lobe, and in the other three the occipital lobe was affected in addition to other parts of the brain. In the first case a small tubercle at the tip of the left occipital pole produced no physical signs during life. In the second case a large glioma involved and destroyed the greater part of the left frontal, temporal and occipital lobes. Partial right homonymous hemianopia was present early in the course of the case. In the third case a gumma was situated on the outer surface of the posterior part of the left occipital lobe. For fifteen years the patient had suffered from epilepsy with an aura of flashes of light before the right eye and followed by blurring of vision for some days. In the fourth case a nodule the size of a large walnut was found on the outer surface of the left occipital lobe just above the pole, with only slight infiltration of the underlying white matter. Fifty times a day the patient had paroxysmal attacks of headache with flashes of light before the right eye, varying in size from that of a button to that of a large globe. This series of cases illustrated the occurrence of tumours of the occipital lobe with partial homonymous hemianopia and without any changes in the visual fields.

Duret [8] discussed the clinical manifestations of tumours of the occipital lobe in detail and classified the clinical features of the condition into four distinct groups. His first group was characterized chiefly by contralateral homonymous hemianopia, in illustration of which were quoted cases reported by Seguin [24], Schoenborn and Weber. In Seguin's case left homonymous hemianopia was the only sign of a tubercle, 2 cm. in diameter, on the calcarine fissure of the right occipital lobe. Schoenborn's case was characterized by crises of headache, impairment of vision and left homonymous hemianopia in association with a cystic sarcoma of the cuneus, precuneus and neighbouring occipital convolutions. On the other hand, Weber's case of sarcoma of the lingual and fusiform lobes was

associated with homonymous hemianopia alone. Duret's second group included those cases of tumour of the occipital lobe which were characterized by hemianopia, hemiplegia and hemianæsthesia ; and was made up of tumours of the inner surface of the occipital lobe penetrating deeply, those of the white matter and those occupying the occipital horn of the lateral ventricle. According to Duret, these physical signs were due to involvement of the adjacent sensory and motor paths in the internal capsule. He quoted cases reported by Jastrowitz, Deschefelder, Jany, Lafforgue, Wordsworth and Spiller in support of his conclusion. His third group consisted of those cases in which homonymous hemianopia was associated with sensory aphasia—word blindness, psychic blindness, agraphia and partial motor aphasia. Duret's fourth group was made up of large tumours which produced all the physical signs mentioned above, and sometimes ocular and facial paralysis in addition.

McKennan [19] described the clinical features of a cyst which approached the outer surface of the right occipital lobe close to the pole. In addition to a history of impairment of health and of epileptiform attacks beginning in the left leg, the patient suffered from word blindness and had left homonymous hemianopia with preservation of central vision for 5° from the fixation point. McKennan summarized the diagnosis, pathological features and results of twenty-four cases of tumour of the occipital lobe which had been reported in the literature, but did not discuss their clinical features.

Zenner [31] reported a case of sarcoma occupying the greater part of the left occipital lobe with complete right homonymous hemianopia up to the fixation point, slight variable weakness of the right arm and leg, difficulty in finding the right word and alexia.

Mills and others [20] mentioned the difficulty of differentiating a tumour of the occipital lobe from one of the cerebellum and quoted Weeks [29], who stated that papillœdema had been found in approximately 87 per cent. of both pre-occipital and cerebellar tumours in a series of 677 cases of cerebral tumour he had reviewed.

Knapp [17] examined the incidence of mental symptoms with cerebral tumours affecting different parts of the brain. Of four cases of occipital tumour mental symptoms had been present in three. He quoted Schuster [23] who had found 4·3 per cent. of occipital tumours among 775 collected cases of cerebral tumour, and mental symptoms in 60 per cent. of those affecting the occipital lobe.

Cushing and his co-workers [6] created renewed interest in the value of the examination of the visual fields in the localization of cerebral

tumours, and in particular differentiated the quadrantic defects in the visual fields occurring with tumours deep in the temporal lobe.

Weisenburg [30] reported a case of tumour of the right occipital lobe, with pressure upon the sensory and motor fibres of the same side and extension into the occipital lobe of the other side. Jamieson [15] had previously reported a case of this type in a patient who was blind, the blindness being regarded as double hemianopia due to involvement of the optic radiations on both sides. In this case the tumour affected first the optic radiation on the left side and later that on the right side. In Weisenburg's case, however, the tumour affected the second optic radiation only partially and so produced a left homonymous hemianopia, together with a right homonymous quadrantic defect affecting only the upper quadrants. The patient also had irritative visual phenomena, at first seeing objects one above the other when they were not so and later seeing objects in the left visual fields jumbled up to look like scenery, glass or a lot of bricks.

Climenko [5] reported a case in which a tumour of the right occipital lobe was associated with a left homonymous quadrantic defect of the visual fields. The patient saw many-coloured animals and objects before his eyes when his headache became severe, and at times saw myriads of rapidly moving objects and various colours. Loud splitting noises like those of a hammer on an anvil were heard when the headache was severe. The left homonymous defect in the visual fields was characterized by blindness of the whole of the upper quadrant and of a small portion of the lower quadrant. In a series of 99 brain tumours recorded in 1916, Clarke [4] found four tumours of the occipital lobe, three of which were gliomata and one an endothelioma.

Bregman and Mesz [2] described a case of angioma of the left occipital region associated with a systolic bruit, marked apertures in the overlying bone on X-ray examination, paresis of the left arm and leg, nystagmus on looking to the left and a nævus of the scalp over the affected area. Puusepp [22] reviewed the clinical features of eight cases of tumour of the occipital lobe, in all of which headache, vomiting, motor and sensory symptoms, papilloedema and hemianopia were present.

Recently, Frazier and Waggoner [9] made the first critical survey of the clinical features of tumours of the occipital lobe in a paper given before the Philadelphia Neurological Society. These writers surveyed forty cases, in nine of which the tumour had invaded the occipital lobe from the adjacent part of the temporal lobe. They found convulsions

in 25 per cent., papilloedema in 67·5 per cent., headache as an important symptom in 90 per cent., and inequality of the pupils with the larger pupil on the contralateral side in 25 per cent. In 7·5 per cent. no definite localizing signs were available, since the tumour involved the convex surface of the occipital lobe, a silent area. In some cases there was a reasonable doubt as to the side on which the tumour was situated apart from the symptoms of involvement of adjacent structures. Of these, signs indicating involvement of the parietal lobe—diminution of stereognostic perception, hemiparesis, hemianæsthesia, deviation of the tongue to one side, and lower facial weakness—were found to be most useful. Aphasia was present in approximately 12·5 per cent. when the tumour was adjacent to or involved the temporal lobe. Symptoms suggesting dysfunction of the frontal lobe, which occurred in a maximum of 10 per cent., were sometimes a source of confusion. Cerebellar symptoms were often conspicuous and were regarded as indicating the presence of a tumour in close relation to the tentorium cerebelli. A staggering gait and nystagmus were found most often in from 10 to 12 per cent., while ataxia, awkward movements of the hands, past-pointing and asynergia were found in only from 2 to 5 per cent. Waggoner regarded hemianopia as the most important sign in localization, and found that none of the cases bore out the conception that hemianopia of occipital origin was less clear-cut than that due to lesions further forward.

The position of the knowledge in regard to the clinical features of occipital tumours as revealed in the literature may therefore be summed up as follows: (1) Symptoms of increased intracranial pressure have been of most value in drawing attention to the condition, while visual hallucinations, both formed and unformed, have been recorded as occurring, though rather infrequently. (2) Distortions of the visual fields have been regarded as the most important evidence of localization except in those cases in which the convex surface of the occipital lobe has been affected. Though unusual types of field defects have been reported in individual cases, dogmatic statements persist in some quarters that homonymous hemianopia up to the fixation point and in others that homonymous hemianopia with spacing of the field for central vision are characteristic of tumours of the occipital lobe. Though complete hemianopia, hemianopia with preservation of the field for central vision, partial hemianopia, and quadrantic defects have all been described in reports of individual cases, little attention has been paid to the possibility of variations in the visual field defects with occipital

tumours. (3) Neighbourhood symptoms and physical signs have recently been regarded as important in the lateral and differential localization of tumours of the occipital lobe.

III.—The Symptoms of Occipital Tumours.

As a rule the symptoms of a focal lesion of the brain may be discussed under the following heads: (1) the symptom or symptoms which first drew attention to the presence of an intracranial condition; (2) the symptoms directly attributable to involvement of the part affected; (3) those due to interference with the functions of adjacent structures; and (4) those which can be attributed to a general increase in intracranial pressure. In studying the symptoms of tumours of the occipital lobe it was soon evident, however, that such a dogmatic classification of symptoms *per se* was not at once possible, as the origin of many of them was more or less obscure. Further, while it seemed possible to arrive at the cause of many symptoms which were of a negative character the sense that they indicated a diminution or abolition of function, it was impossible to determine the source of those which were of a positive character and might have been due as much to release of the function of adjacent or associated parts, or to stimulation of the functions of the parts directly involved.

(1) *The first symptom.*—The symptoms which first drew attention to the presence of an intracranial lesion varied considerably. In approximately 75 per cent. of cases they appeared to be directly attributable to a general increase in intracranial pressure, while in the remaining 25 per cent. they were more or less of a focal nature.

In 30 per cent. of cases the onset of the condition was heralded by an epileptiform attack or by a sudden loss of consciousness, but in only one-third of these cases was the attack preceded by a visual aura. In only one case did visual hallucinations alone draw attention to the condition. In 35 per cent. headache of frontal, occipital or general distribution was the initial symptom. In 17 per cent. symptoms of general mental impairment, such as failure of memory, disorders of conduct, change in character or "neurasthenia," were present for a long time before the presence of an intracranial tumour was suspected. In 15 per cent. a transient loss or progressive failure of vision first drew attention to the condition. In one case a squint was noticed before any other symptom appeared. Aphasia was a prominent early symptom in at least three cases—"an inability to find the right word"—in two cases in association with epileptiform attacks, and in one case with failure of

memory. Apart from the occurrence of a visual aura in 10 per cent. of cases and of visual hallucinations in one case, the initial symptom did not suggest that the occipital lobe might be affected. In fact, throughout, symptoms suggesting involvement of the occipital lobe were comparatively infrequent, and were confined to visual hallucinations and to symptoms suggesting that part of the visual field was blind.

(2) *Visual hallucinations.*—These occurred at some time in the clinical course in 25 per cent. of the cases studied, and in 15 per cent. were the typical "unformed" images of an occipital lesion. In only 10 per cent. of cases did the visual hallucinations represent the aura of an epileptiform attack.

"*Unformed*" *hallucinations* occurred in the following cases. (*a*) The patient saw white lights dancing straight in front of the eyes for ten minutes at a time. An endothelioma was found growing from the falx cerebri, invading the inner aspect of the occipital lobe and damaging the optic radiation anterior to the calcarine cortex. (*b*) For five years the patient, aged 12 years, had suffered from attacks of *petit mal* characterized by a sudden loss of consciousness and associated with a visual aura. Before the attack something seemed to come over the eyes, especially on the right side, and after a short time flashes of light appeared in front of the right eye. At operation a tumour was found deep in the left occipital lobe. (*c*) Twelve months before he was first seen the patient had a bilious attack during which he saw a round ball of bright light in front of the right eye. Since then he had been conscious of a black disc with a light behind it, always moving about in front of the right eye and preventing him seeing things. At operation, a tumour involving the left occipital lobe was exposed and patches were seen coming to the surface in the region of the left supramarginal gyrus. (*d*) The patient had had four major epileptic fits, two of which were preceded by an aura of "flashes of flame" before the eyes. The visual fields were normal. A hard, pearly tumour the size of a golf ball was removed from the outer surface of the right occipital lobe about two inches in front of the occipital pole. (*e*) Epileptic fits occurred six months before the patient was first seen and were preceded by a visual aura. "Flashes of light" were seen from time to time for fourteen days before the first fit. The visual fields were normal. An hydatid cyst was removed from the outer surface of the right occipital lobe behind the supramarginal gyrus. (*f*) Two years before he was first seen the patient suddenly saw a series of flashes which seemed to come from above downwards in the right visual field. The flashes were alternately blue and red, and seemed to

be brilliant though dark. They continued rhythmically and regularly for about fifteen minutes and prevented the patient seeing anything else. They were described as being narrow lines like lightning, but straight. Subsequently these attacks occurred about twice a week for fifteen minutes at a time. At other times the patient was conscious of an object moving up and down to the right of the middle line. A gumma was found infiltrating the inner aspect of the left occipital pole over an area about three centimetres in diameter.

Distortion of visual images occurred in two cases. In the case last mentioned the patient remarked that the edge of the pavement on his right appeared to be converging towards the fence on his left. In a further case there seemed to be a mist with specks in front of the eyes, and at intervals "everything seemed to be long and tall." The visual fields were unaffected. A tumour about the size of a golf ball and firm in consistence was found coming to the surface over an area about two and a half centimetres in diameter on the outer aspect of the left occipital lobe near the pole.

A "formed" visual hallucination occurred in two cases. Auditory hallucinations which sometimes assumed the form of his teacher's voice speaking to the patient were associated with a visual hallucination in the form of the teacher himself. A large cystic glioma was found occupying the anterior part of the occipital lobe and the adjacent part of the temporal lobe. The "formed" hallucination in the second case is described in the paragraph on auditory hallucinations. Occurring in a patient with a tumour of the occipital and temporal lobes it took the form of an old woman with a hood coming towards the patient. It was associated with "formed" auditory hallucinations.

In a further case there was *difficulty in recognizing objects*, an abnormality which might be described more accurately as a failure of picture memory. In addition, however, the patient did not know through which window to look to see objects with which he had been acquainted for many years. As in this case the splenium of the corpus callosum was involved as well as the occipital lobe, it was possible that the interruption of the normal association paths between the two occipital lobes prevented the patient localizing objects satisfactorily.

It is interesting to note that in 75 per cent. of the cases of occipital tumour studied no visual hallucinations occurred at any stage of the clinical course.

In few of the cases without hallucinations the visual fields were

entirely unaffected, though in some there was a mere constriction of the contralateral temporal and of the homolateral nasal field, and in many the field for central vision was unaffected. In those cases in which the tumour was confined to the occipital lobe at or near the pole the visual hallucinations, when present, were always of the "unformed" type, and occurred both in cases in which the visual fields were unaffected and in those in which hemianopia was present either up to the fixation point or with spacing of the field for central vision. Visual hallucinations would appear to have occurred, therefore, both when the physiological continuity of the visual fibres was unaffected and when it was completely interrupted. It is apparent that normal visual paths must have been present to convey stimuli from the level of the lesion to the areas of the cerebral cortex in which the stimuli were interpreted as flashes of light and colour; but that complete physiological continuity of the path from the periphery to the visuo-psychic area was not essential. It was equally apparent, however, that in most cases the physiological continuity of some or all of the visual fibres was unaffected, as was shown by preservation of the greater part of the visual field or of that for central vision only. Hence it might be concluded, as suggested by Harris [12], that in the presence of a lesion affecting the visual path, visual hallucinations are more likely to occur when the central visual apparatus is at the same time exposed to stimuli from the periphery. Stimulation of the visual fibres at the level of the lesion, and preservation of the physiological continuity of the visual path from the level of the lesion to the visuo-psychic area would appear to be the conditions essential for the occurrence of visual hallucinations, and to be more likely to produce them when at the same time the physiological continuity of the visual path from the periphery is more or less preserved. This explanation is the only one which takes account of the occurrence of visual hallucinations with hemianopia up to the fixation point.

Furthermore, the conditions under which visual hallucinations occur: (1) with normal visual fields but with a tumour adjacent to the visual fibres among which changes due to variations in circulation or to œdema are apt to occur; and (2) with a tumour situated at or near the occipital pole and producing complete or partial interruption of the visual path, suggest that comparatively few of the visual fibres may be affected and that the few stimuli arising from the affected fibres must of necessity be interpreted as simple and unformed objects.

The occurrence of formed and complex visual hallucinations in association with a tumour in the anterior part of the occipital lobe and

encroaching on the posterior part of the temporal lobe would likewise appear to depend on the stimulation of numerous visual fibres and on the normal stimuli arising from the periphery. The more numerous and diverse stimuli arising from such a lesion are interpreted with less difficulty as an object with the mental picture of which the patient has had some previous acquaintance.

The occurrence of distortion of visual images in two cases, one in which the lesion probably involved the visuo-sensory area, and the other in which it involved the visuo-psychic area, is of particular significance. It would seem that partial interruption of the visual path at a higher level, whether at the visuo-sensory level or between that and the visuo-psychic level, while permitting a correct interpretation of the nature of the object, may at the same time lead to apparent distortions of shape, size and form. In addition, the actual failure of picture memory when the lesion is situated a little anterior to the visuo-psychic area and in the region of the left supramarginal gyrus, suggests that the latter is the result of a similar interruption of physiological continuity at a slightly higher level in the visuo-sensory system. Though suggestive, the evidence of one case is insufficient to show that defects in the localization of objects which have been seen repeatedly is the result of disturbance of the normal association between the two occipital lobes by a lesion of the splenium of the corpus callosum.

(3) *Symptoms suggesting abnormality of the visual fields.*—As a rule, when the visual fields are affected suddenly, the patient is aware of the defect at once. Such is usually the experience of patients in whom thrombosis of the occipital artery occurs on one or both sides. It was unusual, however, for the patient to be aware of the defect in those cases in which the visual fields were affected as the result of an occipital tumour. In only 16 per cent. could the patient recall an experience which indicated that his visual field was limited on one side. This experience varied in different patients. Usually the patient found himself colliding with people on the affected side and sometimes he collided with objects as he walked round them. In only one case did the patient state definitely that he could not see on the affected side. The patient, who was a golfer, gave a very suggestive history. He collided with objects on his left as he passed them. Also when he pulled a ball at golf it at once disappeared from view, but when he sliced it remained in view for the greater part of its flight. In many cases an obscuring of perception, as a result of increased intracranial pressure, may have explained why the patient was unaware of his defect. In

others it was probable that the defect developed so gradually that he readily adapted himself to it.

The above symptoms represent those which may be fairly attributed to involvement of the occipital lobe. It is obvious that by extension, or by œdema or vascular changes adjacent to it, a tumour of the occipital lobe may result in symptoms which suggest involvement of the temporal or parietal lobes, of the speech centres or of the cerebellum. In addition, the general increase in intracranial pressure resulting from the presence of the tumour may also produce symptoms and physical signs which suggest involvement of structures at some distance from the original lesion. These two groups of symptoms are considered below.

(4) *Epileptiform attacks or loss of consciousness.*—Epileptiform attacks or sudden loss of consciousness occurred in 52·5 per cent. of the cases studied, and in 30 per cent. were the symptoms which first drew attention to the presence of an intracranial lesion. In only 10 per cent., however, were the attacks preceded by a visual aura. Epileptic attacks, therefore, occurred much more frequently in this series of tumours of the occipital lobe than in an average series of tumours of the brain.

In order to find if there was any direct relationship between the position of the tumour and the incidence of epileptiform attacks, those cases in which they occurred were classified according to the parts involved. Of the twenty-four cases in which the tumour was confined to the occipital lobe, epileptic attacks occurred in only 8 cases; while of the ten cases in which the temporal lobe was involved as well attacks occurred in five cases: and of the six cases in which the parietal lobe was involved as well attacks occurred in five cases. These figures suggested that epileptic attacks were probably less frequent in those cases in which the tumour was confined to the occipital lobe, and relatively more frequent if the temporal or parietal lobe was involved as well.

The relationship of the occurrence of epileptic attacks to the presence of increased intracranial pressure at the time they occurred demands some consideration. Accepting headache, vomiting, vertigo and papill-œdema as a fair indication of the presence of increased intracranial pressure, it was found that in very few cases were the attacks related to the presence of increased intracranial pressure which had become definitely established. More often, however, they were related to sudden crises of headache, vomiting and vertigo which were presumably manifestations of a temporary increase in intracranial tension. In such cases the attacks were often followed by weakness of one or more limbs,

which either persisted or recovered in a few days. Occasionally an epileptiform attack of a "focal" type occurred, but that was the exception.

It was unusual for the attacks to be numerous. In only two cases did the clinical picture assume the characteristics of "idiopathic epilepsy," in one case for two years and in the other for five years, before other symptoms and physical signs appeared. More often other symptoms appeared within a few weeks and left no doubt that the attacks had an organic basis. As a rule, apart from the two cases mentioned, the patient had from one to six attacks before coming under observation.

In spite of the apparent influence of the focal lesion, œdema, vascular changes and temporary or permanently increased intracranial pressure upon the occurrence of epileptiform attacks, it was evident that some factor other than focal irritation or increased intracranial tension must have played a part in many cases. It many cases in which the lesion was equally of a focal type or the intracranial pressure was raised no such attacks occurred.

The details of the attacks themselves were difficult to determine, as in most cases it was necessary to depend upon relatives for a description. Often, however, it was possible to obtain sufficient information upon which to identify the nature of the attacks. They were of the following types: (*a*) sudden loss of consciousness and a fall without any convulsions; (*b*) epileptiform attacks of a Jacksonian type; (*c*) major epileptiform attacks with general convulsions; and (*d*) attacks of *petit mal* with or without a visual aura.

Occasionally the attacks were preceded by an aura—visual, auditory or sensory, and sometimes a feeling of cramp in one limb. The occurrence of a visual aura usually indicated that some part of the visual path was involved, probably in the occipital lobe, but did not suggest that the tumour itself had involved the visual fibres. The occurrence of a "formed" visual hallucination before a fit suggested involvement of the anterior part of the occipital lobe or extension into the temporal lobe. Unformed auditory or sensory hallucinations, such as noises in the ear, numbness or tingling, occurred sometimes when the temporal or parietal lobes were invaded, but more often when the tumour was confined to the occipital lobe. They were thus of no value in detailed localization. Formed auditory hallucinations, however, taking the form of voices speaking and in some cases conveying definite information, occurred only when the anterior part of the occipital lobe and the

adjacent part of the temporal lobe were involved. Unformed auditory and sensory aura, local cramps in one limb, and both transient and permanent weakness of an arm and a leg after an attack, occurred in cases in which the tumour was entirely confined to the occipital lobe, and did not necessarily indicate that the tumour had invaded the temporal or the parietal lobe.

(5) *Auditory hallucinations.*—Noises were heard by the patient, usually before a fit, in two cases. In one case they preceded the onset of symptoms suggesting a transient increase in intracranial pressure. Sometimes they were like the buzzing of bees or of voices, but often they formed themselves into the voice of the patient's teacher speaking to him. The teacher's words seemed to be a repetition of something the patient had heard him say, but the actual words were never heard. A formed visual hallucination of the figure of the teacher was sometimes associated with the auditory hallucination. The tumour was found to involve the anterior part of the occipital lobe and the posterior part of the temporal lobe.

In the second case the auditory and visual hallucinations were described by the patient as follows : " When I have the heavy feeling coming over me, I see gold stars falling in front of me. They are in front and not to one side. Then I see sort of wings flapping. Also I hear a sort of 'ha-ha'ing in the ears ; and sometimes a voice, a strange voice like nothing earthly, saying, ' Why have you not told me before ? Why did you not tell me long ago ?' Sometimes I see an old woman with a hood coming towards me. Then I feel a strange heavy sinking sensation in the pit of the stomach. Then I lose myself." In this case the tumour was found occupying the adjacent parts of the occipital and temporal lobes.

(6) *Headache.*—Headache was present in 95 per cent. of the cases studied and in 35 per cent. was the first symptom. In practically every case it was associated with other symptoms and physical findings indicative of increased intracranial pressure. In two cases, however, it was absent, though a moderate degree of papilloedema was present in each case. No characteristic distribution of the headache was noted. While usually frontal or vertical in distribution, it sometimes involved the occipital region as well. Occurring at first at infrequent intervals, usually on arising in the morning and improving later in the day, it became gradually more frequent and severe, and was associated with nausea, vomiting and giddiness. Finally, in some cases it awakened the patient during the night and persisted for as long as five days at a time.

Acute crises of increased intracranial pressure characterized by severe headache, vomiting and vertigo, sometimes occurred at long intervals before the headache became permanent. Rarely, an attack of headache occurred in which the distribution was entirely occipital. As was to be expected, however, when the headache was almost always associated with increased intracranial pressure, its distribution gave no clue whatever to the site of the lesion.

(7) *Vomiting.*—Vomiting was usually absent in the early stages of a case of occipital tumour, even when severe morning headaches occurred. However, on the headache becoming more severe and prolonged, it was usually associated with nausea. Eventually vomiting occurred during such attacks. It occurred often at the height of the headache, sometimes once on getting out of bed and sometimes only once or twice during the whole course of the illness. In about 20 per cent. of cases vomiting did not occur, though a moderate or severe degree of papilloedema was present.

(8) *Vertigo.*—Vertigo was an infrequent symptom even during the crises of increased intracranial pressure characterized by severe attacks of headache and sometimes by vomiting. "A swimming in the head" was more usual. The feeling that objects were moving round was not experienced, nor was a sensation of falling in one particular direction. Occasionally the patient tended to go to one side or the other, a tendency which was satisfactorily explained by disturbances of motor function on one side of the body.

(9) *Diplopia.*—Diplopia, occurring either intermittently or for a month or two at a time, was not common. It occurred in only 22 per cent. of the cases studied. The duplication of images usually occurred on lateral deviation of the eyeballs, was associated with a slight defect of lateral deviation to one or both sides, and sometimes with slight irregular nystagmoid jerkings of the eyeballs on lateral deviation. When present diplopia was invariably associated with an increase in intracranial pressure. In at least nine cases in which there was a definite increase in pressure no history of diplopia was elicited.

(10) *Impairment of vision.*—Impairment of vision appeared as a definite symptom in 57 per cent. of cases and was the initial symptom in 15 per cent. Occasionally the failure of vision was intermittent, objects being dark before one or both eyes for a day or two at a time. Such intermittent impairment of vision was particularly apt to occur during the crises of increased intracranial pressure. It was a common statement of the patient that objects looked at appeared to be dim for

an hour or two after an attack of headache. In one case dimness of vision and what was practically blindness appeared after seeing a series of bright lights. In other cases difficulty in seeing letters and words was noticed early in the history of the condition, the difficulty becoming more pronounced as the condition progressed. More often the patient complained of a mist with specks in front of the eyes. These variations in vision were almost invariably associated with papillœdema, and sometimes when progressive with the development of secondary atrophic changes in the optic disc.

(11) *Abnormal tastes and smells.*—The patient complained of abnormal tastes and smells either alone or before a fit in five cases, in three of which the tumour was confined to the occipital lobe, and in two of which the tumour involved the temporal lobe as well. In no case could the taste and smell be identified, though in two cases a resemblance was noticed between the taste or smell and that of something of which the patient had had previous experience. One patient in whom there was a tumour towards the external surface of the left occipital and temporal lobes was for two months conscious of a peculiar smell which he failed to identify. Another patient occasionally had a taste like blood in the mouth, and here again the tumour involved the left occipital and part of the left temporal lobe. In the remaining three cases in which the tumour was confined to the occipital lobe the smell or taste was described as unpleasant but could not be recognized.

(12) *Disturbances of the speech functions.*—A history of disturbance of the speech functions was particularly valuable in the localization of a tumour affecting or spreading to the outer surface of the left occipital lobe. The speech functions were affected in 35 per cent. of the cases studied. When the parietal lobe was affected in addition to the occipital lobe, or the tumour extended to the outer surface of the occipital lobe, the speech defect was more or less permanent. A transient attack of motor aphasia or difficulty in finding the right word was common for a short time after an epileptiform attack.

The commonest defect was failure of picture memory, either of individuals, objects, words or letters: and frequently one function alone was affected. In 25 per cent. there was alexia or failure of picture memory. In four of these cases there was also difficulty in finding the right word, and in several others in which the left occipital lobe was involved there was transient difficulty in finding the right word during acute crises of increased intracranial pressure. As a rule the patient complained that he could not read words or letters, but one patient, a

cartoonist, was known to have been making his figures without arms or legs. The details of the disturbances of speech functions and their relation to the pathological changes present are dealt with in the discussion of the physical signs.

(13) *Subjective sensations.*—Spontaneous subjective sensations were present in 30 per cent. of cases; were associated with involvement of the occipital lobe alone in 10 per cent.; of the occipital and temporal lobe as well in 10 per cent.; and of the occipital and the parietal lobe as well in 10 per cent. The subjective sensations occurring with tumours of the occipital lobe alone were a feeling of numbness, coldness or tingling in the contralateral arm or leg. In one case there were spasmodic attacks of pain in the right side of the face, which probably had no direct relation to the tumour itself. In one case in which the tumour extended into the temporal lobe there was a constant feeling of cold and shivering all over the body.

(14) *Disturbances of motor functions.*—In some cases the nature of the disturbances of motor function as described in the history suggested lesions of motor fibres, and in others disturbances of cerebellar function. They were present in 52 per cent. of the cases studied.

In 40 per cent. of the cases the symptoms suggested involvement of the motor fibres. In some they appeared suddenly after an epileptiform attack, and in others they came on gradually in the later stages of the illness. One patient in whom the tumour had extended slightly into the left parietal lobe had noticed that the right leg was dragging for three weeks In a second patient the left arm and leg became weak after two epileptiform attacks two months before admission. She complained that she dropped things from the left hand and dragged the left foot when walking. In a third patient similar symptoms appeared after an epileptiform attack, and in a fourth patient they developed gradually. In these cases the symptoms suggesting involvement of the motor fibres were comparatively indefinite. In no case did the frank symptoms of a hemiplegia appear.

In 18 per cent. of cases the tumour had extended into the adjacent parietal lobe but never far enough to damage the motor fibres by direct involvement. In about half of these cases spontaneous subjective sensations were present as well as weakness of the contralateral limbs. In the remaining 22 per cent. the tumour was confined to the occipital lobe and in about three-quarters of these cases spontaneous subjective sensations occurred in addition to the motor weakness.

In 12 per cent. of cases the history of motor weakness suggested dis-

turbance of the cerebellar functions. Symptoms of this type were a tendency to fall backwards or to one side, weakness of the arm and leg often on the same side as the tumour, and difficulty in controlling the movements of the limbs.

(15) *Disturbances of the sphincters.*—Difficulty in controlling the sphincters was not a characteristic feature of occipital tumours *per se*. Incontinence of urine and fæces occurred at some stages of the clinical course in 12 per cent. of cases. As a rule, it was present during acute crises of increased intracranial pressure and disappeared as soon as the patient recovered from them. In one case it appeared during the late stages of the illness but disappeared when the intracranial pressure was relieved by surgical measures. Incontinence occurred, however, in only a few of the cases in which the intracranial pressure was much raised. In spite of this the evidence would appear to show that rapid increase in the intracranial pressure was the important factor in the production of incontinence, and that the situation of the tumour and other changes in the brain bore no relation to it.

(16) *Mental symptoms.*—Mental symptoms of varying severity appeared in 55 per cent. of the cases studied, and in 17 per cent. were the clinical features which first drew attention to the illness.

As the latter were often of considerable importance in differential diagnosis they are described in detail. In Case 3 the patient's memory for recent events was affected first, but this was soon followed by difficulty in reading and inability to find the right word. In Case 7 the patient felt for a long time that he was getting run down, he was tremulous and had no confidence in himself. In Case 10 the patient was easily excited, lost his temper too easily, and found that his memory for recent events was failing rapidly. In Case 16 the patient had been thought for many months to be suffering from neurasthenia. In Case 32 the patient noticed a general failure of health and felt that he wanted to get away from everything. He often forgot where he was going. In Case 25 the patient had suffered from neurasthenia for sixteen months and was sure that his brain was going. In Case 37 the patient had felt tired for five years, and for three years had shown a definite change in character.

Most of the mental symptoms which occurred in the early stages of the illness were of the psycho-neurotic type as if the patient were less able to adapt himself to his environment and to meet his difficulties. For a time they appeared to justify a diagnosis of neurasthenia.

The mental symptoms occurring during and after the onset of acute

crises of increased intracranial pressure varied considerably. In the simpler cases the patient was merely drowsy and confused. In more pronounced cases he became comatose for a time, and on recovering was confused for an appreciable period. During a crisis one patient was incoherent in speech and failed to recognize his relatives. Another patient was drowsy during the attacks of headache and became more drowsy with successive attacks. In all such cases the mental symptoms were intermittent, the patient returning to a more or less normal degree of mental health between the attacks.

In the more slowly progressive cases the mental symptoms were as follows: Impairment of recent memory, absent-mindedness, excitability, irritability, lack of confidence, inability to think clearly, feeling weak and tired, restlessness, suspicion and making peculiar remarks. These symptoms were of considerable importance. They often occurred before the onset of frank symptoms of increased intracranial pressure when their appearance might easily have led to the suspicion that the tumour had originated in the frontal lobe. Even when they occurred after symptoms of increased intracranial pressure had appeared, they might have been regarded as of the same significance.

IV.—The Physical Signs of Occipital Tumours.

It is most convenient to discuss the physical signs found in association with tumours of the occipital lobe in the order in which they are elicited during the routine examination of the patient. The term "physical sign" is used here to include all evidences of abnormality elicited during the examination of the patient.

(1) *Mental changes*—These were found on examination in 60 per cent. of the cases studied. In every case they were associated with increased intracranial pressure at the time the examination was made. There were, however, several cases with increased intracranial pressure in which no definite psychological abnormalities were found. Some degree of mental impairment was present in all the above 60 per cent., if the ability of the patient to meet the situations of his everyday life were taken as the standard of normality.

The commonest defects were defects of attention and memory, comparatively slight and appearing only under the influence of stress or fatigue in the slighter cases, but very pronounced and constant in the severe cases. In 17 per cent. the patient was unable to fix his attention for any appreciable time; in 7 per cent. he failed to co-operate satisfactorily during examination; and in 12 per cent. cerebration was definitely

slow. In at least 22 per cent. there was definite clouding of consciousness with drowsiness and lethargy. Insight was definitely impaired in one case only.

Attention was impaired in more than half the cases in that the patient had to have his attention repeatedly drawn to a statement or a command; while in two cases the patient had to be roused from his lethargic condition and spoken to repeatedly before he could appreciate what was expected of him.

Orientation was normal in most cases, but in 10 per cent. there was definite disorientation both as to time and place. In one other case the patient was correctly orientated as to time and place but failed to decide whether he was to the left or right of a fireplace near him. In a further case the patient was unable to find his way about in places he knew well, and did not know through which window to look to see objects he had known for many years. As suggested above, these apparent disorders of orientation probably have a more local significance than the disorientation as to time and place which occurs with mental impairment.

Memory was affected in 32 per cent. of cases. As a rule the patient was unable to remember the details of incidents which had occurred a few minutes before; but in 12 per cent. with definite impairment of cerebration there was also mental confusion and loss of memory for remote events. At the same time, the time relations of both recent and remote events were much confused.

Reasoning power was definitely impaired in two cases and speech was incoherent or irrelevant apart from aphasia in three cases. As a rule the patient was emotionally colourless and detached, occasionally he was worried about his condition and his difficulties with speech, while in one case he was definitely facile and euphoric.

Hallucinations and illusions which could be ascribed to mental changes rather than to interference by organic conditions with visual, auditory and sensory paths were practically unknown. One patient, however, regarded his doctor as a policeman, while another became exceedingly suspicious and antagonistic towards those with whom he had been on good terms. In only one case did the hallucination based upon the organic lesions lead to the development of delusions.

(2) *Variations in the sense of smell.*—In 12 per cent. of cases there was difficulty in recognizing the smell of test substances such as cloves, peppermint, lavender and paraldehyde. In one case the presence of nasal catarrh was enough to account for the impairment of smell; while

in three others the mental condition was much impaired at the time the patient was examined. In these three cases the patient was unable to recognize such test substances as cloves and peppermint, but knew that lavender and paraldehyde were different. It was apparent that minor differences were not recognized, whereas definite distinctions were appreciated though not by name. In a further case the sense of smell was completely lost in the left nostril but normal in the right nostril.

(3) *Variations in the visual fields.*—Detailed charts of the visual fields were available in 80 per cent. of the cases studied. In the remaining 20 per cent. it was found impossible to chart the fields because the patient was mentally impaired or was unable to co-operate well enough during the examination. In these cases, however, it was often possible to determine roughly the type of defect in the visual fields.

The following conclusions are based only on those cases in which a detailed record of the visual fields was available, though the percentages given are calculated from the total number of cases studied. In every case but two there was some degree of homonymous hemianopia on the side opposite to that on which the tumour was situated. In individual cases, however, the defect in the visual fields varied considerably with the position of the tumour.

Classified according to the type of the defect in the visual fields, the cases studied were grouped as follows:—

(*a*) Those with complete contralateral homonymous hemianopia extending right up to the fixation point (32·5 per cent.).

(*b*) Those with incomplete contralateral homonymous hemianopia extending to the middle line above and below the field for central vision but leaving the latter more or less unaffected (22·5 per cent.).

(*c*) Those in which there was a contralateral homonymous crescentic defect of the visual fields (7·5 per cent.).

(*d*) Those in which there was a contralateral homonymous quadrantic defect of the visual fields (12·5 per cent.)

(*e*) Those in which the visual fields were normal (5 per cent.).

In addition, there was often superimposed on these types of fields a certain degree of concentric constriction in those cases in which papilloedema had been present for some time or in which optic atrophy was beginning.

(*a*) *Contralateral homonymous hemianopia up to the fixation point.*—This type of defect in the visual fields was found in 32·5 per cent. of cases; and in 5 per cent. of cases unformed visual hallucinations had occurred before and up to the time the visual field defect was discovered.

The temporal and nasal fields were definitely separated by a line which passed directly through the fixation point. The affected temporal and nasal fields were completely blind, while the corresponding nasal and temporal fields were either normal or showed a degree of peripheral constriction which depended upon the condition of the optic discs and the visual acuity.

The only points of importance in the pathological findings were that the position of the tumour was usually such that all of the visual fibres would have been involved by it, and that the proportion of cases in which degenerative changes had occurred was unusually high. Complete interruption of all the visual fibres was essential for the production of this type of field defect.

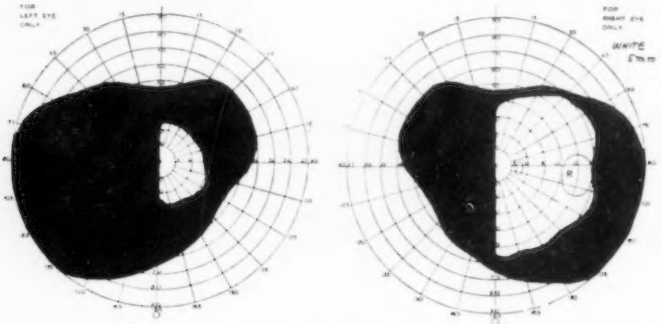

FIG. 1.—Contralateral homonymous hemianopia up to the fixation point due to an endothelioma weighing 135 grammes invading the right occipital lobe. (Case 10.)

(*b*) *Incomplete contralateral homonymous hemianopia with spacing of the field for central vision.*—This type of field defect occurred in 22·5 per cent. of cases and in only one case was associated with a history of unformed visual hallucinations. Above and below the field for central vision the nasal and temporal fields were more or less definitely separated by a vertical line through the fixation point of the normal field, though in some cases the dividing line extended a little towards the blind side. In all cases the field for central vision was more or less unaffected. In some there was definite and regular spacing at ten degrees from the fixation point, but in most cases the field for central vision was not so regularly and completely defined as it was in those cases in which thrombosis of the posterior cerebral artery had occurred in a comparatively young individual. In most cases the spacing of the field

for central vision was irregular and varied from 5 to 10 degrees from the fixation point. In the majority of these cases the tumour extended chiefly towards the external surface of the occipital lobe, while in one case it occupied a position almost entirely internal to the ventricle.

(c) *Contralateral homonymous crescentic defect of the visual fields.*—This type of field defect occurred in 7·5 per cent. of cases. The homolateral halves of the visual fields were normal or only slightly reduced as a part of the general constriction of the visual fields associated with long-standing papilloedema or commencing optic atrophy. The contralateral halves of the visual fields were limited only on the corresponding side of a line drawn vertically through the fixation point. Beginning at the vertical line mentioned, the periphery of the field for peripheral

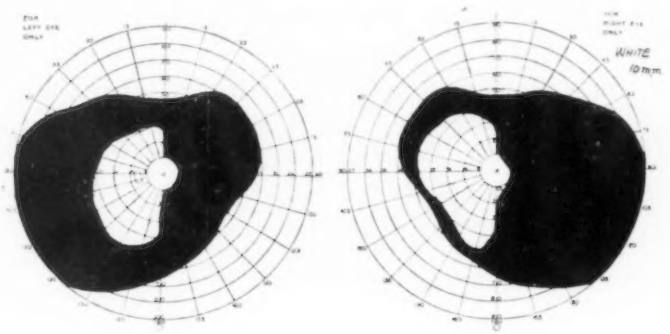

FIG. 2.—Contralateral homonymous hemianopia with sparing of the field for central vision, due to a tumour invading the mesial aspect of the left occipital lobe. (Case 20.)

vision was gradually reduced more and more as far as the horizontal limit of the upper quadrant, and then less and less until it again became normal at the vertical limit of the lower quadrant. In the affected temporal field the defect assumed the form of a broad crescent, extending from the periphery inwards, while in the affected nasal field it assumed the form of a narrower crescent at the periphery. Measured horizontally from the fixation point towards the periphery, the proportion of the temporal field affected was approximately the same as that of the nasal field affected as long as the vision and the condition of the fundi were the same in the two eyes.

The cases in which this type of field defect occurred appeared to have had in common the fact that the tumour in each case approached the optic radiation from the external surface of the occipital lobe.

Though there was insufficient evidence upon which to base any general conclusion, the association of a tumour in that position with a crescentic field defect was worthy of note.

(*d*) *Homonymous quadrantic defect of the visual fields.*—This type of defect of the visual fields occurred in 12·5 per cent. of cases. One quadrant of the contralateral temporal and the corresponding quadrant of the homolateral nasal field were completely blind at the periphery and the defect extended for a varying distance towards the fixation point.

A quadrantic field defect was present in two cases in which the tumour was situated in the anterior part of the occipital lobe and encroached upon the adjacent part of the temporal lobe; in two cases when the occipital lobe alone was affected, and in one case when part of

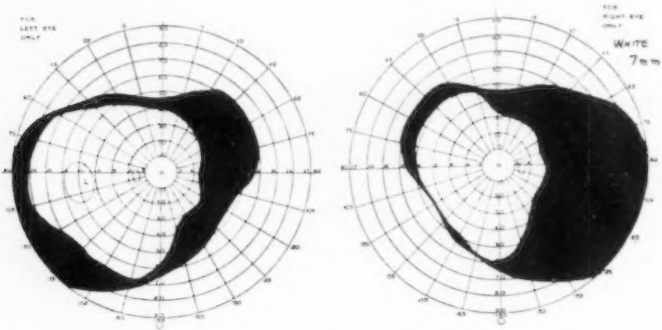

FIG. 3.—Contralateral homonymous crescentic defect of the visual fields due to a tumour invading the convex surface of the left occipital lobe. (Case 14.)

the visuo-sensory area was probably affected. The following are summaries of the pathological conditions present, together with a description of the particular type of field defect found in each case.

(i) A large glioma was found lying close beneath the cortex and occupying the whole of the posterior part of the left temporal lobe and the anterior part of the left occipital lobe. The lower right temporal quadrant showed a visual defect extending to within 30 degrees of the fixation point, while in the left lower nasal quadrant the defect extended right up to the fixation point. (Case 1.)

(ii) An endothelioma growing from the falx cerebri invaded the internal surface of the left occipital lobe about midway between its superior and inferior limits. Antero-posteriorly its position corresponded to a point about midway between the occipital pole and the posterior limb of the

Sylvian fissure. The right upper temporal quadrant showed a defect extending to within five degrees of the fixation point, while the left upper nasal quadrant showed a similar defect extending somewhat nearer to the fixation point. The greater part of the field for central vision was spared on each side. After the tumour was removed there developed a right homonymous hemianopia in which the greater part of the field for central vision was spared. (Case 12.)

(iii) A gumma invaded the inner aspect of the left occipital pole of over an area about three centimetres in diameter. There was general constriction of the visual fields and both the right lower temporal quadrant and the left lower nasal quadrant showed a defect limited to about the lower half of the quadrant and approaching to within five degrees of the fixation point. (Case 40.)

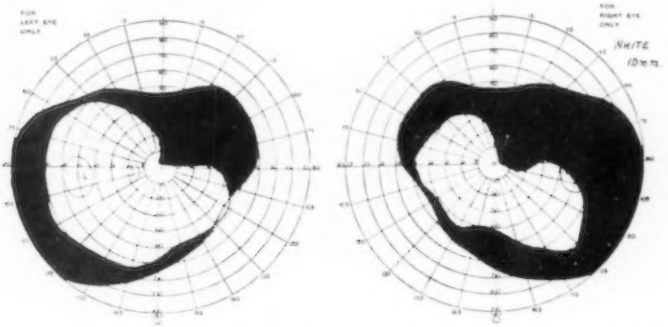

FIG. 4.—Contralateral homonymous quadrantic defect of the visual fields due to an endothelioma invading the mesial aspect of the left occipital lobe just anterior to the calcarine cortex. (Case 12.)

In each of these cases it was apparent that the tumour affected only a portion of the visual fibres or, as in the last case, of the visuo-sensory cortex. In two of the cases the tumour was situated well forward in the occipital lobe, when it might be expected that the defects in the visual fields would be similar to those usually found with tumours of the temporal lobe. Persistent homonymous quadrantic defects of the visual fields are encountered frequently even in the later stages of tumours of the temporal lobe, but are found less often with tumours of the occipital lobe. It is possible, however, that if patients suffering from a tumour of the occipital lobe were studied carefully from the early stage of the illness quadrantic defects of the visual fields would be found more often. Nevertheless, it is important to note that quadrantic defects of the visual fields can and do occur when only a portion of the visual fibres

are invaded by the tumour or even when a tumour invades only a part of the visuo-sensory cortex in the neighbourhood of the calcarine fissure.

(*e*) *Normal visual fields.*—The visual fields were found to be normal in 5 per cent. of cases. In each case the tumour involved the external surface of the occipital lobe. The following are the pathological findings in two cases of this type.

(i) From the outer surface of the right occipital lobe less than two inches in front of the occipital pole a hard pearly tumour about the size of a golf ball was removed. On section the tumour was found to be a gumma. A further examination of the patient four months after the operation showed that the visual fields were still normal. (Case 36.)

(ii) A cyst was removed from the outer surface of the right occipital lobe just behind the supra-marginal gyrus. Microscopical examination showed that the cyst contained a scolex with the characteristic features of *Tænia echinococcus*. (Case 38.)

In both of these cases the tumour was small, was confined to the outer surface of the right occipital lobe and apparently was associated with few changes in the neighbouring brain tissue. In each case it was only the history of a visual aura which made localization and treatment possible.

(4) *Changes in the fundi.*—In 65 per cent. of the cases studied, papillœdema was present, and in 45 per cent. the degree of swelling was over three dioptres. In 20 per cent. the discs were normal, in a further 10 per cent. they were doubtful but probably normal, while in the remaining 5 per cent. atrophic changes had already supervened. In Frazier and Waggoner's series of cases of occipital tumour papillœdema was present in 67·5 per cent., and in the majority of cases there were between three and five dioptres of swelling.

Frequently vision was normal, or only a little diminished, even in the presence of a high degree of papillœdema. When, however, the intracranial pressure had been increased for some time vision was definitely impaired, even in the absence of definite local evidence of atrophy of the optic discs. When atrophic changes were already present in the disc vision was often reduced to the perception of fingers only.

Retinal hæmorrhages were present only with the higher degrees of papillœdema. Occasionally, when the swelling of the discs was pronounced the development of a macular fan was observed. No evidence whatever was found to support the suggestion that retinal hæmorrhages were particularly apt to occur when the increase in intracranial pressure was the result of a tumour of the occipital lobe. The degree of pressure

and the obstruction of retinal veins in their course behind the retina appeared to be the important factors in the incidence of retinal hæmorrhages.

(5) *The pupils.*—In the majority of cases the pupils were equal and reacted normally, whether the light stimulus were applied to the normal or to the blind side of the visual field. Occasionally they were dilated and reacted sluggishly, findings usually associated with some impairment of vision. Occasionally one or both pupils were slightly irregular in outline or eccentric, but nothing was found to account for these variations.

In 35 per cent. of cases the pupils were unequal, and in 25 per cent. the larger pupil was on the same side as the hemianopia or other visual field defect. In the remaining 10 per cent. the smaller pupil was on the same side as the visual field defect. Frazier and Waggoner found the pupils unequal, with the larger pupil on the contralateral side, in 22·5 per cent. of their cases. In no case in either series was the difference in the size of the pupils extreme.

Study of the vision in relation to this inequality of the pupils showed that there was no variation which could account for the contralateral pupil being the larger.

The fact that in 22·5 per cent. of Frazier and Waggoner's cases and in 25 per cent. of this series the contralateral pupil was the larger, suggested that there might be some direct relationship between the nature of the inequality of the pupils and the side on which the tumour was situated. However, the incidence of 10 per cent. of cases in this series in which the homolateral pupil was the larger had to be explained. No local conditions such as unilateral failure of vision or atrophy of the optic disc were found to account for the dilatation of the homolateral pupil. Consequently, it was impossible to arrive at any general conclusion other than that in two-thirds of the cases in which the pupils were unequal the larger pupil was on the contralateral side in relation to the position of the tumour, or on the same side as the visual field defect.

(6) *Ocular pareses and diplopia.*—Disturbances of ocular movements occurred in 30 per cent. of cases.

Most of the changes were those associated with paresis of the abducent nerve on one or both sides. As a rule the disturbances were slight and revealed themselves as a difficulty in deviating the eyeballs to one side or the other, or in irregular nystagmoid jerkings of the eyeballs on attempts at lateral deviation towards the affected side. During acute crises of increased intracranial tension, or late in the course of the case when intracranial tension was increased, definite internal strabismus was

apparent. As a rule, the patient complained of diplopia, especially on lateral deviation during the acute phase, but did not do so during the chronic phase. These changes occurred in 17·5 per cent. of the cases. Since they were most pronounced during acute crises of increased pressure or in the long-standing cases in which the intracranial pressure was raised, it is probable that they were the direct result of the increased pressure upon the long and exposed course of the sixth nerve on each side.

In two cases some degree of bilateral ptosis was evident. In one case, though papillœdema was slight, the patient was drowsy and muddled and was constantly making an attempt to break through the fogged state in which he found himself (Case 13). In the second case there was a high degree of papillœdema and probably hydrocephalus.

In Case 18 there was difficulty in closing one eye on account of weakness of that side of the face; in Case 26 there was proptosis of the left eye; and in Case 27 there was a divergent strabismus of the left eye.

(7) *Nystagmus and nystagmoid jerkings.*—Jerking movements on lateral deviation of the eyeballs were found in 35 per cent. of the cases studied. These movements were of two distinct types: (*a*) nystagmoid jerkings; and (*b*) a varying degree of true nystagmus.

(*a*) Nystagmoid jerkings occurred in 15 per cent. Fine horizontal movements, often irregular both in time and amplitude, occurred on lateral deviation of the eyeballs. Though sometimes present on lateral deviation to one side and not to the other, or more pronounced to one side and not to the other, they bore the same general characteristics throughout. They were associated in each case with a high degree of papillœdema and therefore with a pronounced increase of intracranial pressure. They were probably due to the difficulty of maintaining the lateral position of the eyeballs owing to lack of balance of the ocular muscles following pressure on one or both of the abducent nerves. The group of cases in which they occurred did not necessarily coincide with that in which the patient complained of diplopia or in which an internal strabismus was evident on lateral deviation. In no sense could these nystagmoid jerkings be ascribed to direct or indirect interference with cerebellar function.

(*b*) Movements which were very suggestive of true nystagmus occurred in 20 per cent. of cases. Because of the frequently quoted difficulty, in the absence of charts of the visual fields, of differentiating a tumour of the occipital lobe from one of the lateral lobe of the

cerebellum on the same side, it was necessary to consider these cases in detail.

(i) In Case 8 there was rhythmic, horizontal and slow nystagmus on looking to the extreme right, and similar movements but of smaller amplitude on looking to the left. Associated features of the case were a high degree of papillœdema, definite paresis of the abducent nerve on each side and general muscular hypotonia more pronounced on the left than on the right side, though on the latter the tone was increased somewhat by a lesion of the motor fibres. A large perivascular sarcoma was found occupying the greater part of the left occipital lobe.

(ii) In Case 9 lateral deviation of the eyeballs to the left was accompanied by rhythmical horizontal nystagmus and that to the right by less marked nystagmoid jerks. Both discs were normal. The tumour involved the right occipital lobe.

(iii) In Case 10, on lateral deviation of the eyeballs to the right there were nystagmoid jerks of wide amplitude, while to the left there were a few fine jerks. In walking the patient showed a constant tendency to stagger to the right. A large endothelioma weighing 135 grm. occupied the right occipital lobe.

(iv) In Case 21 there was slight, horizontal nystagmus on looking to the right and the left—rapid, fine, irregular and ill-sustained. There was general hypotonia, especially on the right side, and inco-ordination with the finger-nose test, especially in the right hand. There was an extreme degree of papillœdema. The tumour occupied the left occipital lobe.

(v) In Case 23 there was regular nystagmus on looking to the left. A moderate degree of papillœdema was present. An ill-defined soft tumour was found about 1 in. below the outer surface of the right occipital lobe.

(vi) In Case 28 there was well-marked nystagmus at the rate of about five jerks per second on looking to the right. There were five dioptres of papillœdema in each eye. The tumour involved the left occipital lobe.

(vii) In Case 33 slow nystagmus was present with coarse movements on looking both to the right and to the left. A large thin-walled cyst occupied the right occipital lobe.

It was evident that in some cases the presence of nystagmus could be explained by cerebellar dysfunction secondary to increased intracranial pressure, the nystagmus and hypotonia being false localizing signs due to transmission of the high supratentorial pressure on to the

cerebellum. In some cases direct local pressure on the cerebellum with or without increased intracranial pressure was responsible; in others a contrecoup pressure upon the contralateral lobe of the cerebellum may have occurred; while in others again no satisfactory explanation could be found to account for the presence of nystagmus. Distortion of the brain-stem owing to increased general pressure in addition to the lateral pressure may have accounted for some of the more obscure examples.

The following conclusions appeared to be justified on the evidence available in this series.

(*a*) Movements of the eyeballs resembling true nystagmus were present in 35 per cent. of the cases of tumour of the occipital lobe.

(*b*) In 15 per cent. of cases there was a mild degree of abducent paralysis on one or both sides which, on extreme lateral deviation of the eyeballs and the attempt to maintain the lateral position of the eyeball, resulted in nystagmoid jerkings.

(*c*) In 20 per cent. of cases there was nystagmus which suggested the presence of a cerebellar lesion—horizontal, slow and of full amplitude on lateral deviation to one side, and fast and of smaller amplitude on lateral deviation to the other side. In most of these cases the signs appeared after the onset of symptoms of increased intracranial pressure, and were the result of cerebellar dysfunction secondary to that condition. In the minority of these cases nystagmus was present throughout the clinical course and was associated with other symptoms which suggested a lesion of the cerebellum on the side of the nystagmus of greater amplitude. In these cases the findings were the result of direct or contrecoup pressure of the tumour locally on one lateral lobe of the cerebellum, and were probably aggravated by the presence of increased intracranial pressure.

(8) *Variations in the functions of the trigeminal nerve.*—Abnormalities of function of one or both trigeminal nerves were found in 25 per cent. of the cases studied. The changes varied from sluggishness of one corneal reflex and slight impairment of light tactile sensation on one side of the face to a definite impairment of tactile and pain sensation. In no case was sensation completely lost. It was more usual to find a distinct difference between the two sides with only moderate impairment of the finer variations on the affected side. As a rule, such changes were associated with similar changes in the limbs and trunk on the same side, but occasionally when the variations in sensation were slight the distribution of the trigeminal nerve alone was affected. In such cases a clear demarcation of the abnormal from the normal was

impossible. In only 7·5 per cent. did the tumour involve the parietal lobe at all; in 2·5 per cent. it had involved the temporal lobe; and in 15 per cent. it was entirely confined to the occipital lobe. In only one case was anæsthesia in the distribution of the trigeminal nerve associated with a spontaneous sensation of numbness.

(9) *Facial paresis.*—A suggestion of weakness of one side of the face was a common finding, and occurred in 57·5 per cent. of the cases under review. In no case, however, was a definite facial paralysis of either the peripheral or the central type found. In every case the suggestion of facial weakness was confined to the side opposite to that on which the tumour was situated. In 40 per cent. the weakness of the face was associated with indefinite or definite weakness of the arm and leg on the same side, and in several cases with abnormalities of sensation in the arm and leg on the same side. In others there was weakness of the arm and leg without any weakness of the face.

Such variations in facial weakness were comparatively slight—a difference in the naso-labial folds, a smoothing of one side of the face, or a slight lagging of one side of the face on voluntary movement. In others the face was smoother and flatter on one side than on the other, and the palpebral aperture on that side was narrower than normal. In others definite weakness of one side of the face at the angle of the mouth was evident on the patient showing the teeth or closing the eyes at command.

In 17·5 per cent. of cases, however, the emotional movements were affected as much as or more than the voluntary movements. The following are illustrative cases.

(1) In Case 3 there was slight weakness of the right side of the face on attempting to smile. On testing the facial muscles for voluntary movements, however, there was much less obvious weakness of the lower part of the right side of the face. The facial folds were greater on the right side of the face, but there were more wrinkles on the left side of the forehead. Associated with these findings there were also alexia, weakness of the right arm and leg, dragging of the right foot and a history of pins and needles, tingling and numbness in the right hand. The tumour occupied the left occipital lobe and encroached somewhat on the parietal and temporal lobes as well.

(2) In Case 9 there was slight weakness of the left side of the face only on emotional movement, together with weakness of the left arm and leg, and impairment of the sense of passive movement in the left arm and leg. The tumour was confined to the right occipital lobe.

In these cases the variations in the facial movements, whether confined to emotional movements alone or to emotional and voluntary movements together, were associated with evidence of involvement of the motor fibres in the corresponding arm and leg. It would appear, however, that when the involvement of the motor fibres is very slight the weakness of the face may be appreciated only on involuntary and emotional movements and give the impression that the paths for emotional movements are involved rather than those for voluntary movements. The conscious effort needed to initiate voluntary movements is enough to overcome the very slight weakness due to involvement of motor fibres so rapidly that delay in movement is not appreciated by the observer, whereas with involuntary movements some delay is at once noticeable. This observation does not necessarily contradict the belief that emotional movements are merely controlled by the more direct motor paths.

(10) *Involvement of other cranial nerves.*—The impairment of hearing which occurred in four cases was probably independent of the intracranial condition. In no case were the glosso-pharyngeal, vagus or spinal accessory nerves affected. A slight deviation of the tongue to one side was observed in some cases, but it was probably more apparent than real, as it was associated with slight weakness of the same side of the face and of the limbs on the same side.

(11) *Variations in the speech functions.*—Abnormalities of the speech functions were found on examination in 30 per cent. of the cases studied, and in 50 per cent. of those in which the left cerebral hemisphere was affected. No speech defect other than a transient difficulty was found in cases in which the right cerebral hemisphere was involved. Aphasia of some degree was present in 33 per cent. of cases in which the tumour was confined to the left occipital lobe, in 71 per cent. of cases in which the left parietal lobe was affected as well, and in 60 per cent. of cases in which both the left temporal and occipital lobes were involved. When the tumour definitely involved or approached the external surface of the left cerebral hemisphere, aphasia was present in 66 per cent., while in the remaining cases in which the tumour was confined to the middle or inner part of the occipital lobe, aphasia occurred in only 9 per cent. It seemed, therefore, that either the speech areas or their connections had to be directly invaded by the tumour, or by œdema or vascular changes close to it, before permanent defects of speech appeared.

Nominal aphasia was found in 20 per cent., or in 33 per cent. of left-

sided tumours; some form of visual agnosia in 22·5 per cent., or in 37·5 per cent. of left-sided tumours; and auditory agnosia in only one case. Other minor changes were found but were of little significance compared with the above abnormalities.

The types of speech defect found and their relation to the position of the tumour are illustrated by the following cases.

(i) In Case 3 the patient carried out movements correctly at command, but did not always obey the command at once. From time to time he perseverated. He read 11·53 as "seven min twel." He made no attempt to read or to spell words. When asked to name a series of objects he made fruitless attempts to recall the names. He knew the uses of objects and when he heard their names said "Yes" at once. A large tumour was removed from the outer aspect of the left occipital lobe and was found to involve the adjacent parts of the parietal and temporal lobes.

(ii) In Case 14 the patient had to be helped if he could not find the word he wanted. He could not name objects but could touch an individual object if it were named. He had some difficulty in reading written words. He could pick out objects when they were named and could write the names to dictation. A tumour the size of a hen's egg was removed from the subcortical part of the left occipital lobe close to the temporal lobe.

The presence of some degree of alexia was of considerable value in the localization of those occipital tumours which approached the external surface of the left occipital lobe. It was often associated with nominal aphasia.

(12) *Disturbances of sensation.*—Variations in sensation were present in 55 per cent. of cases. They were observed on one side of the body, not only in those cases in which the tumour had encroached on the part of the parietal lobe adjacent to the occipital lobe, but also in cases in which the tumour was confined entirely to the occipital lobe. They were, however, more common in those cases in which the tumour partly encroached upon the parietal or the temporal lobe. In every case they were confined to the side of the body opposite to that on which the tumour was situated. In some cases they were found over the face, arm, trunk and leg, while in others they were more or less confined to or more pronounced in the face, arm or leg. Occasionally, because of the impaired mental condition of the patient or of his difficulty in expressing himself, it was impossible to determine the degree of sensory impairment other than the changes in the cruder sensations.

Minor variations in sensation were the rule, while complete loss of sensation was not observed. Tactile stimuli and moderate differences in painful stimuli and between heat and cold were appreciated better on one side than on the other. In some cases these forms of sensation were merely impaired on one side when compared with the other, while in others there was a distinct difference in the responses when the two sides were stimulated. Various degrees of difficulty in appreciating the shape, size and weight of objects were observed. One patient was able to appreciate the weight of objects but could not be sure of their size. Slight difficulties were more common than complete loss of the power to distinguish the shape, size and weight of objects, though the latter did occur. Two-point discrimination and localization were impaired in all degrees, from a greater proportion of errors than on the other side of the body to complete loss of the power to localize a painful stimulus accurately. The sense of position and of passive movement were impaired in some cases, while in others there was impairment and sometimes loss of the power to appreciate vibrations when a tuning-fork was applied to the radius or malleolus.

When the appreciation of touch, pain and temperature were slightly impaired, the sense of position, two-point discrimination, localization and the appreciation of the shape, size and weight of objects were usually impaired as well. When, however, the latter group only were impaired, some were often affected and others normal. For example, in Case 25, in which the tumour was confined to the right occipital lobe, the patient had slight difficulty only in appreciating changes in position and passive movement in the left arm. In Case 26, in which the tumour involved the left occipital lobe and part of the parietal lobe, there was only some loss of the sense of position in the right arm. In Case 27, where the tumour affected the right temporal as well as the occipital lobe, the localization of painful stimuli was less accurate in the left arm than in the right arm.

In every case minor degrees of variation in tactile, pain and thermal sensations only were found, and in most cases localization, two-point discrimination, appreciation of slight changes in the position of the finger and toe, and the appreciation of variations in the size, shape and weight of objects were impaired rather than abolished.

The changes found were essentially those of involvement of the sensory paths above the level of the optic thalamus. With them was found in some cases difficulty in appreciating finer variations when a tuning-fork was applied to a bony prominence.

(13) *Motor abnormalities in the limbs.*—Motor abnormalities were found in the arm and leg in 90 per cent. of the cases studied. Minor degrees of muscular weakness, of alterations in muscle tone and of variations in the reflexes were the rule. It was difficult in many cases to decide what was the origin of the motor abnormality, and it was only after careful consideration of the distribution of the disability, the motor changes, the variations in co-ordination and the changes in the reflexes that an opinion could be expressed as to the nature of the motor change. In every case the following probable causes were considered : (*a*) Involvement of the motor fibres ; (*b*) disturbance of cerebellar function on one side from direct pressure upon one lateral lobe of the cerebellum ; (*c*) bilateral cerebellar dysfunction from a general increase in intracranial pressure ; and (*d*) motor weakness from loss of sensation.

The comparative unimportance of cerebellar dysfunction from direct pressure as a cause of unilateral motor weakness was suggested by the fact that in practically every case the limbs affected were on the side opposite to that occupied by the tumour. Bilateral diminution of muscle tone with motor weakness and diminution of the tendon reflexes occurred in some cases in which the intracranial pressure was much increased. When such changes appeared after evidence of involvement of the motor fibres was already present, it was often found that the tendon reflexes on one side were absent while those on the other side were sluggish, the general diminution in the tendon reflexes probably being the result of the increased intracranial pressure and the sluggishness of the reflexes on one side due to the fact that the already exaggerated reflexes on that side persisted under such conditions. In other cases, definite variations in muscle tone and motor power, exaggeration of the tendon reflexes, diminution or absence of the abdominal reflexes and a doubtful or definite movement of dorsiflexion with the plantar reflex, left no doubt that the motor disability was the result of interference with the motor fibres. Occasionally a case was encountered in which no direct evidence of interference with the motor fibres or with the cerebellum was found to account for weakness of the contralateral arm and leg, whereas definite interference with sensory functions was evident and appeared to provide a satisfactory explanation for the weakness of the limbs and the difficulty the patient had in using them. While loss or diminution of tactile, pain and thermal sensation sometimes made the limbs clumsy and weak, it was usually when the sense of position was affected that the weakness of the limbs was most evident. This occurrence of motor disability with disturbance of sensation was

regarded as analogous with that found when thrombosis of a branch of the middle cerebral artery had affected the posterior limb of the internal capsule, in which condition the resulting motor disability was often more disturbing to the patient than that associated with interference with motor fibres.

The above factors were evidently responsible for the motor disability in individual cases, and in some cases two or more of the factors were jointly responsible for the weakness of the limbs. The changes described in the following illustrative cases were characteristic of those occurring with tumours of the occipital lobe.

(i) In Case 2 the outstretched left arm tended to assume an abnormal attitude and fell away. The grip of the left hand was slow and weak. The finer movements of the fingers of the left hand were clumsy. The fingers missed an object more often with the left than with the right hand. There was slight weakness of the left leg compared with the right. Co-ordination was poor in both hands. On the left side the cremasteric reflex was sluggish and the plantar reflex was extensor. There was a well-marked flexion reflex on the left side. A tumour the size of a golf ball was removed from the upper and anterior part of the right occipital lobe. It reached the middle line and involved the adjacent part of the parietal lobe. In this case there was definite evidence of slight involvement of the motor fibres, probably by vascular changes or œdema in the neighbourhood of the tumour.

(ii) In Case 9 the left arm fell away on extension and was feebler than the right arm at every joint. There was sensory ataxia of the left arm. Hypotonia of the left arm was evident on passive movement and on hyper-extension. In the left leg tone appeared to be increased peripherally and there was inco-ordination with the heel-knee test. The tendon reflexes on the left side were slightly more active than were those on the right side. The sense of passive movement was impaired in the fingers and toes on the left side and later hypotonia was more pronounced in the left arm. The tumour occupied the right occipital lobe. In this case the extreme weakness and difficulty in using the left arm and leg could be explained satisfactorily by loss of the sense of position in those limbs and by the presence of sensory ataxia.

(iii) In Case 8 there was weakness of the lower half of the right side of the face for voluntary and emotional movements. There was slight weakness and increase in muscle tone in the right arm and leg. The tendon reflexes on the right side were more active than those on the left side, and the right plantar reflex was extensor. In walking, the

patient dragged the right leg and failed to swing the right arm. Later there was definite nystagmus of wider excursion and more pronounced on lateral deviation of the eyeballs to the left. There was also severe hypotonia of the left arm, together with some decrease in tone in the right arm. The tumour occupied the left occipital lobe and was accompanied by a high degree of papillœdema.

In this case it was difficult to assess the relative importance of the different factors combining to produce the variations in motor function. At first there was evidence in the changes in the right arm and leg of interference with motor fibres, but the subsequent appearance of nystagmus and hypotonia on the left side was more difficult to explain. They may have been due to the increase in intracranial pressure, the muscle tone being reduced less in the right arm and leg because of the previous increase in tone due to the motor lesion. It was more probable that local pressure by the tumour on the left lobe of the cerebellum was aggravated by the general increase in intracranial pressure, and the preponderance of what were more or less definite signs of a cerebellar lesion on the left side produced in that way.

(iv) There were cases, however, in which the increase in intracranial pressure was apparently responsible for general muscular hypotonia and weakness, and even for the loss of tendon reflexes. Of this, Case 1 was an example. The knee-jerks were absent, while the right ankle-jerk was absent and the left present. Both plantar reflexes were flexor. Later, there was weakness of the right side of the face, and the right plantar reflex was sometimes extensor. During generalized convulsions the left arm and leg moved more than the right. There were seven dioptres of papillœdema in the right eye and six dioptres in the left. A large tumour occupied the anterior part of the right occipital lobe and the adjacent part of the temporal lobe. Here the general muscular hypotonia and the loss of tendon reflexes were probably the result of increase in the intracranial pressure and the persistence of a tendon reflex on one side due to a relative increase in tone on that side from slight involvement of the motor fibres.

From the cases examined it was evident that there was some difficulty in interpreting the disturbances of motor function associated with tumours of the occipital lobe. The motor changes were often slight, but, nevertheless, were often of much value in deciding upon which side the tumour was situated. There were, however, serious limits to their value, as was shown by the following case.

The patient, female, aged 19, was seen on February 22, 1930. She

had had epileptiform attacks for nine years at intervals of about three weeks. These attacks were preceded by a strange feeling over the front of the head and in the epigastrium, but no visual symptoms were present. Twelve months before she was seen she had had "a nervous breakdown." On examination, the following positive findings were noted. The patient was mentally alert and co-operative, but obviously fatigued easily during the examination. Lateral deviation of the eyeballs to the right was defective, and attempts to deviate to the left resulted in a few irregular nystagmoid jerks. There was a definite left homonymous hemianopia with apparent spacing of the field for central vision. There were six dioptres of papilloedema in each eye. The right angle of the mouth was below the left at rest, and definite weakness of the lower part of the right face was evident on showing the teeth at command. There was a slight decrease in motor power and tone in the right arm compared with the left; and the motor power in the right leg was decreased compared with that of the left. In the arms the tendon reflexes were more active on the right side, the right abdominal reflex tired and disappeared before the left, and the right plantar reflex was probably flexor while the left was definitely so. There was a slight defect of localization of painful stimuli in the right arm compared with the left. There was a fine tremor of the fingers and repeated alternating movements were more awkward with the right arm than with the left.

In this case the signs in the right side of the face and in the right arm and leg suggested interference with the motor fibres in the left side of the brain, and it was only the definite left homonymous hemianopia which made it possible to localize the lesion in the right occipital lobe. The motor signs were probably false localizing signs which had developed after and because the intracranial pressure had been increased. At operation a large cyst was removed from the outer aspect of the right occipital lobe.

On the material studied the following conclusions as to the incidence and nature of motor changes in association with tumours of the occipital lobe appeared to be warranted.

(*a*) Minor disturbances of motor function occurred in practically all cases of tumour of the occipital lobe.

(*b*) The majority of them occurred in the contralateral limbs and as a rule were found to be due to involvement of the motor fibres by œdema or other changes in the tissues adjacent to the tumour. Changes due to involvement of motor fibres could, however, occur in the homo-

laterel limbs as false localizing signs due to and following an increase in intracranial pressure.

(*c*) Occasionally motor weakness in the contralateral limbs was associated with and probably due to loss of the sense of position.

(*d*) In some cases tumours of the occipital lobe were associated with general muscular hypotonia and motor weakness, probably because of of cerebellar dysfunction following the rise in the supratentorial pressure.

(*e*) In cases in which the factor mentioned above came into play the persistence of the reflexes in the contralateral limbs was probably due to interference with the motor fibres in the neighbourhood of the tumour.

(*f*) True cerebellar dysfunction affecting the arm and leg on one side was unusual, and when present may have been due to local pressure by the tumour or to local pressure aggravated by the increase in supratentorial pressure. Rarely there was a suggestion of cerebellar dysfunction affecting the contralateral limbs, which may have been due to a contrecoup pressure disturbing the contralateral lobe of the cerebellum.

V.—Discussion.

Many of the clinical features of tumours of the occipital lobe have been discussed as they arose in the course of this paper, but certain general features of these lesions still remain to be considered.

The clinical course of the condition varied considerably. In some cases epileptiform attacks continued for as long as eight years before there arose symptoms or signs which more directly suggested the presence of an intracranial condition. In others, in approximately 57 per cent., general mental impairment, headache and transient or progressive failure of vision—symptoms suggesting a general increase in intracranial pressure—appeared early and the case ran its course in a few months. In these cases there was little doubt that the condition was symptomless until the onset of symptoms of increased intracranial pressure, a feature of more than half of the cases which made it very difficult to bring them under treatment before the more serious risks of increased intracranial pressure arose. The early appearance of symptoms suggesting the involvement of visual paths was unusual. Visual hallucinations occurred alone in only 2·5 per cent. and as the aura of an epileptiform attack in only 10 per cent. Epileptiform attacks appeared to be the most valuable of the initial symptoms in drawing attention to

the presence of an intracranial condition as they occurred first in 30 per cent. of cases, in one-third of them with a visual aura and often as a few isolated attacks. It was apparent, therefore, that in only 10 per cent. of cases was it probable and in only 30 per cent. was it possible, to have the patient brought under treatment before the risks of increased intracranial pressure were incurred, and then only if a detailed examination of the nervous system were carried out.

When the patient came under observation the frequency of symptoms directly suggesting involvement of the visual paths was very little increased. By that time epileptiform attacks had occurred in 52·5 per cent., an unusually high figure compared with the average of 10 per cent. in an unselected series of intracranial tumours. The high incidence of other symptoms of increased intracranial pressure in association with such attacks—headache in 95 per cent., diplopia in 22 per cent., impairment of vision in 57 per cent., and disturbance of the sphincters in 12 per cent.—suggested that increased intracranial pressure was a factor of the greatest importance in the high incidence of epileptiform attacks. The higher incidence of epileptiform attacks in those cases in which the tumour had invaded the parietal or the temporal lobe as well as the occipital lobe appeared to be of importance only in so far as the extension of the tumour into those parts conditioned the occurrence of particular types of aura and the distribution of the convulsive movements.

Symptoms suggesting involvement of the central visual paths were limited to two—visual hallucinations and experiences suggesting abnormality of the visual fields. Visual hallucinations occurred in only 25 per cent. of cases. In 15 per cent. they were of an unformed type, which appeared to be due to an incomplete lesion close to the visual area of the cortex and such as might have resulted from early invasion of the optic radiations by tumour, by œdema or by vascular changes. Formed images, which occurred in 5 per cent., appeared to be the result of similar incomplete involvement of the visual paths in the anterior part of the occipital lobe and in the temporal lobe, where it was possible for a relatively large number of scattered fibres to be affected in such a way that the resulting stimuli were interpreted without difficulty as being visual stimuli arising through normal channels from formed objects external to the individual. Unformed hallucinations, therefore, seemed to be due to a lesion in the posterior part of the occipital lobe, while formed hallucinations suggested that the lesion was confined to the anterior part of the occipital lobe or had invaded the adjacent part of the

temporal lobe. When sufficient information was forthcoming hallucinations were found to occur in the contralateral hemianopic field of vision, and were thus of some value in confirming the localization of the lesion when it affected the convex surface of the occipital lobe, especially on the right side in those cases in which no other definite localizing signs were found. In those cases in which the phenomenon occurred distortion of visual images was of some value in localizing the tumour in that part of the convex surface of the occipital lobe just anterior to the occipital pole. Experiences suggesting abnormality of the visual fields were rarely volunteered, but when described might have been of some help in determining the side upon which the tumour was situated in those cases in which it was no longer possible to confirm the information so gained by charting the visual fields.

Physical signs indicating involvement of central visual paths were confined to the information gained by charting the visual fields. It was possible, however, for a tumour of the convex surface of either the right or the left occipital lobe to be present without any distortion of the visual fields. When affecting the latter, disturbances of speech were usually of value in determining the position of the tumour, but when affecting the former it was possible for no localizing sign to be present. Actually, however, distortion of visual images owing to involvement of the visuo-psychic area or a history of unformed visual hallucinations due to the extension of œdema or vascular changes into the adjacent optic radiations, strongly suggested that in such cases the tumour affected the convex surface of the occipital lobe and in the absence of disturbances of speech favoured a localization in the convex surface of the right occipital lobe. No cases of tumour affecting the internal surface of the occipital lobe were found without some alteration of the visual fields, probably because the presence of the visuo-sensory area in the calcarine fissure and the inward course of the optic radiation towards this part and to the occipital pole made it improbable that a tumour could be for long in that region without interrupting the visual path.

A contralateral homonymous crescentic defect of the periphery of the visual fields, mentioned by Traquair [27] and suggested in one case by Bramwell [1], appeared to be confined to those cases in which the tumour was invading the occipital lobe from its convex surface. The essential feature of this type of visual field defect was the proportionate contraction of the contralateral temporal and of the homolateral nasal fields. It is probable that a tumour approaching the optic radiation from the outer aspect would affect first the fibres midway between the uppermost

and the lowermost, and as it extended further interrupt more fibres above and below that level. That this type of field defect was produced in that way was suggested not only by the occurrence of cases in which only a peripheral crescentic contraction of the field was found, and of others in which the crescentic contraction of the field extended to within 20° of the fixation point, but also by the repeated observation of cases in which the contralateral temporal field became more and more contracted at successive examinations.

On the contrary, a tumour growing from the region of the falx cerebri into the internal surface of the occipital lobe appeared to have a greater chance of producing a contralateral homonymous quadrantic defect of the visual fields, especially if it were situated towards the upper or lower limit of the optic radiation. If only the upper or lower part of the visuo-sensory cortex in the calcarine fissure were involved early by a small lesion, a contralateral homonymous quadrantic defect of the visual fields would result. Invasion of the optic radiation slightly anterior to the visuo-sensory area of the cortex occurred in several cases and resulted in a similar homonymous field defect. In Weisenburg's [30] case a tumour invading one occipital lobe from the other in its anterior part produced homonymous hemianopia on one side and an homonymous quadrantic defect on the other side. Such a defect was possible if only the upper or lower fibres of the optic radiation were affected where they were arranged in a somewhat crescentic manner around the outer wall of the lateral ventricle. Similarly, a tumour deep in the anterior part of the occipital lobe could invade the lowermost fibres of the optic radiation as they passed upwards and backwards from the temporal loop and produce a contralateral homonymous quadrantic defect. The importance of these findings is the recognition of the fact that tumours affecting the lips of the calcarine fissure, those invading the internal surface of the occipital lobe just anterior to the calcarine fissure, and those deep in the anterior part of the occipital lobe can produce a contralateral homonymous quadrantic defect of the visual fields.

When the tumour arose in the part through which the optic radiation passed or extensively involved the optic radiation secondarily, a contralateral homonymous hemianopic defect of the visual fields with preservation of the field for central vision was found. It appeared that as long as a certain minimal number of visual fibres failed to be destroyed and the occipital cortex in the neighbourhood of the occipital pole was preserved, the field for central vision was unaffected. It

seemed that for this to occur some stimuli for the cortical area for central vision in the neighbourhood of the occipital pole must have been conveyed by every corticopedal fibre in the optic radiation. It was possible, however, for the lesion to have destroyed those fibres passing to the calcarine cortex for peripheral vision and to have left those passing to the occipital pole unaffected.

Tumours large enough to have involved the greater part of the occipital lobe, those undergoing cystic degeneration, and those which infiltrated extensively the substance of the occipital lobe, produced a complete contralateral homonymous hemianopic defect up to the fixation point. In such cases, all of the fibres must have been interrupted.

The defects in the visual fields found in association with a tumour of the occipital lobe were thus shown to vary considerably with the position of the tumour and the degree to which it affected the fibres of the optic radiation. Quadrantic defects, usually described as characteristic of tumours of the temporal lobe, were equally probable with small tumours involving the calcarine fissure, and with large tumours invading the optic radiation from the inner aspect, or from below when situated in the anterior part of the occipital lobe. A tumour could be on the convex surface of the occipital lobe for a long time without producing any alteration in the visual fields, and one invading the optic radiation from the outer aspect could produce a slowly developing contralateral crescentic defect of the periphery of the visual fields. The frequent occurrence of a contralateral homonymous hemianopic defect with spacing of the field for central vision appeared to be due to preservation of fibres conveying stimuli for central vision, which may have been simply preservation of any of the fibres of the optic radiation; to destruction of those passing to the calcarine cortex and preservation of those passing to the occipital pole; or to destruction of the calcarine cortex and preservation of that at the occipital pole, together with preservation of many of the fibres of the optic radiation. A complete homonymous hemianopic defect up to the fixation point appeared to be due to destruction of all the fibres of the optic radiation; to destruction of the calcarine cortex and of the fibres passing to the occipital pole; or to destruction of both the calcarine cortex and that at the occipital pole with or without destruction of all the fibres in the optic radiation.

Neighbourhood symptoms and signs were found to be of considerable importance in cases of occipital tumour, not only in confirming

the diagnosis of a focal lesion, but also in the detailed localization of the tumour. Only in the case of speech defects, however, did it appear to be necessary for the tumour to directly invade the adjacent parts, in order to produce the characteristic clinical features of a lesion of that part. Disturbances of the speech functions occurred in 50 per cent. of the cases in which the left occipital lobe was involved, and in 66 per cent. of the cases in which the tumour approached or involved the external surface of the lobe. Nominal aphasia, visual agnosia, and especially alexia, were of value in the localization of a tumour which had approached the external surface of the left occipital lobe, and in the majority of cases indicated definitely that the tumour was closer to the external surface than to the internal surface. Other neighbourhood symptoms and physical signs did not appear to have the same significance. Auditory hallucinations of the formed type and abnormal tastes and smells, which as a rule could not be identified, appeared to indicate disturbance of the functions of the temporal lobe, but owing to the position of the tumour in the cases in which they occurred, could not have been due to direct invasion of that lobe. Similarly, spontaneous subjective sensations in 30 per cent. of cases, and disturbances of motor function in 52 per cent., could have been the result in only a few cases of direct invasion of the parts by the tumour. While direct invasion of the central sensory paths by the tumour might have been responsible for spontaneous subjective sensations such as tingling, numbness and pins and needles in a few cases, in the majority they could not have been the result of direct involvement. In any case, this factor could not have accounted for the history of motor disabilities. When the results of physical examination were considered as well, it was found that sensory disturbances of a suprathalamic type and minor degrees of motor disturbances in the face, arm and leg of the opposite side were exceedingly common, the former in 52 per cent. and the latter in 90 per cent. The mild degree of these disturbances, their appearance in many cases shortly after symptoms of increased intracranial pressure had arisen, and the fact that they probably indicated involvement of structures in direct relation to the tumour itself, suggested that they were the result of œdema or vascular changes in the cortical and subcortical tissues of the post-central and pre-central parts of the parietal lobe on the same side as that on which the tumour was situated. Similarly, the symptoms already mentioned as indicating involvement of the temporal lobe were probably the result of the same mechanism.

Neighbourhood symptoms and signs of a different type were those

which seemed to suggest involvement of one lobe of the cerebellum. While comparatively infrequent, giddiness, hypotonia in the homolateral limbs and nystagmus were common enough to be considered in the differential diagnosis of the condition. Though comparatively slight and indefinite in the presence of normal intracranial pressure, they appeared to be much aggravated when the intracranial pressure was increased. Such findings were probably the result of the direct transmission of pressure on to the lateral lobe of the cerebellum on the same side and were probably more definite when the intracranial pressure was raised because the increased supratentorial pressure transmitted through the tumour on to the lateral lobe of the cerebellum produced much more definite disturbances of cerebellar function.

Many of the clinical findings, however, which were likely to cause difficulty in diagnosis and localization were probably the direct or indirect results of increased intracranial pressure, whether due to secondary hydrocephalus or to œdema in the tissues adjacent to the tumour. General hypotonia and diminution of the tendon reflexes were probably the result of the direct transmission of the supratentorial pressure on to the cerebellum as a whole, while the apparent diminution in the muscle tone, motor power and reflexes on the homolateral side was often the result of the same mechanism, the difference in the findings on the two sides being due to involvement of motor fibres on the same side as the tumour. Similarly, indefinite nystagmus may have been due to the same mechanism or to mild degrees of weakness of one or both external recti muscles. Headache, papilloedema and transient or progressive failure of vision were definitely the result of increased pressure, while transient loss of vision in the hemianopic field was more probably the result of invasion of the central visual paths by œdema in the tissues adjacent to the tumour.

The disturbances of mental functions which were present in many cases were probably the result of one or both of two factors. In many they were undoubtedly the result of the disturbance of cerebral functions consequent upon the raised intracranial pressure and were more likely to appear when the pressure was raised rapidly; but in other cases it was evident that the presence of a tumour even in the occipital lobe was sufficient to disturb the normal cerebral associations enough to produce disturbances of memory and attention and to cause the patient to develop neurasthenic symptoms because of his impaired reaction to his environment. That such a factor as the latter came into operation in patients with tumours of the occipital lobe was of some importance

when it was considered that such symptoms were often believed to suggest primary involvement of the frontal lobe by a tumour. That being so, early disturbances of mental function and neurasthenia might have been regarded as false localizing signs.

False localizing signs which actually led to an erroneous localization of the lesion were comparatively infrequent in the cases studied. It was apparent, however, that with tumours involving the convex surface of the occipital lobe and under conditions in which it was impossible to chart the visual fields there were many symptoms and signs which might have led to an incorrect localization. Symptoms and signs suggesting disturbance of cerebellar function and those indicating disturbances of mental function have already been mentioned. Formed visual hallucinations and abnormal tastes and smells might have suggested a lesion of the temporal lobe, spontaneous subjective sensations and disturbances of sensation of the suprathalamic type, a lesion of the post-central part of the parietal lobe, and minor degrees of motor dysfunction, a lesion of the precentral area, or of the corresponding frontal lobe when associated with even slight mental changes. In all such cases the chronological order of the appearance of the symptoms and physical signs was of supreme importance, if it were possible to determine it in detail, as it often was. Disturbances which appeared before the occurrence of one or more symptoms suggesting increased intracranial pressure were more likely to be due to a lesion of the part concerned with the functions disturbed, while those which appeared later were likely to be the result of the increased pressure. The appearance of symptoms suggesting a lesion of the temporal or of the parietal lobe at the same time as or shortly after symptoms of increased pressure might have been due to the extension of œdema about the tumour. If the increased pressure were due to secondary hydrocephalus, as it was in some cases, it was possible for the false localizing signs which developed subsequent to the rise in pressure to indicate a localization on the side opposite to that occupied by the tumour. This did not occur in any of the cases studied, but did occur in a case already quoted to illustrate this point. In this case the localizing signs mentioned appeared to indicate a lesion of the left parietal lobe, whereas the discovery of left hemianopia at an earlier stage and the displacement of a calcified area in the falx cerebri towards the left, showed that the actual lesion was not only in the right cerebral hemisphere but in the right occipital lobe. This opinion was confirmed at operation.

The relative importance of definite localizing signs, neighbourhood symptoms and signs, and the symptoms and signs of increased intracranial pressure in cases of tumour of the occipital lobe appeared to depend upon the correct appreciation of the time relations of the symptoms and signs, and upon the relative severity of the signs elicited. Distortion of the visual fields appeared to be the only sign upon which reliance could be placed and then only when incomplete or complete homonymous hemianopia was found. A contralateral homonymous crescentic defect of the visual fields was useful in localizing a tumour of the convex surface of the right occipital lobe, and appeared to be a sign worth searching for repeatedly in those cases in which symptoms of an intracranial lesion were present and no definite clue could be obtained to its situation. Neighbourhood symptoms and signs were of considerable importance in localizing the lesions, while those due to increased pressure were apt to be confusing and to lead to an erroneous localization if their true significance were not appreciated.

Apart from the differential diagnosis of tumours of the occipital lobe from tumours in other parts of the brain, the actual diagnosis of the presence of a tumour was often a matter of some difficulty. In many cases in which epileptiform attacks were a prominent feature, a detailed examination of the nervous system revealed findings which made it clear at once that an intracranial tumour was present. There were others, however, in which epileptiform attacks had occurred for some time and physical examination revealed no definite indication of the presence of a focal lesion. The only procedure possible in such cases was to regard the condition provisionally as idiopathic epilepsy and to re-examine the patient at intervals in the search for signs of a focal lesion. In this connection the examination of the optic discs and the charting of the visual fields appeared to be the most important.

The differential diagnosis from vascular conditions was never easy in the absence of unequivocal evidence of increased intracranial pressure. The difficulty was not, however, so common as might have been suggested by the absence of papilloedema or of other changes in the optic discs in 30 per cent. of cases. The diagnosis of a focal lesion and often the localization of the lesion itself were possible on the physical signs alone, while the slowly progressive nature of the illness with occasional exacerbations of headache, nausea, impairment of vision and clouding of consciousness appeared to offer direct evidence of a neoplastic rather than of a vascular lesion. Nevertheless, certain sources of confusion were evident. Homonymous hemianopia with spacing of the field for

central vision could have been produced with equal facility by thrombosis of the posterior cerebral artery and by a tumour interrupting the visual path. Here again the manner of onset and the clinical history offered data upon which a differentiation could be made.

VI.—Summary and Conclusions.

(1) The clinical features of 40 cases of tumour of the occipital lobe have been reviewed, and their frequency, pathogenesis and significance discussed.

(2) The initial symptoms were epileptiform attacks in 30 per cent.; a visual aura or visual hallucinations in 12·5 per cent.; general mental impairment in 17 per cent.; headache in 35 per cent.; transient or progressive failure of vision in 15 per cent.; and strabismus in 2·5 per cent. In only 12·5 per cent. did the initial symptoms suggest that the visual paths were involved.

(3) The relative frequency of different symptoms of tumour of the occipital lobe was as follows: Visual hallucinations, 25 per cent.; symptoms suggesting abnormality of the visual fields, 16 per cent.; epileptiform attacks, 52·5 per cent.; auditory hallucinations, 5 per cent.; abnormal tastes and smells, 12·5 per cent.; headache, 95 per cent.; diplopia, 22 per cent.; impairment of vision, 57 per cent.; disturbances of speech, 35 per cent.; spontaneous subjective sensations, 30 per cent.; disturbances of motor functions, 52 per cent.; and mental symptoms, 55 per cent.

(4) Important findings on the examination of the patient were the following: Mental changes, 60 per cent.; contralateral homonymous defects of the visual fields, 94 per cent.; papillœdema or optic atrophy, 70 per cent.; inequality of the pupils, 35 per cent. (contralateral pupil the larger in 25 per cent.); ocular pareses, usually of the external recti, 30 per cent.; nystagmus and nystagmoid jerkings, 35 per cent.; disturbances of speech functions, 30 per cent. (50 per cent. of left-sided tumours); disturbances of sensation of the suprathalamic type, 55 per cent.; and minor degrees of motor disturbance usually in the contralateral limbs, 90 per cent.

(5) The changes in the visual fields found in the majority of cases occurred only in the contralateral halves of the fields. In individual cases the visual fields showed: (*a*) complete hemianopia up to the fixation point; (*b*) incomplete hemianopia with spacing of the field for central vision; (*c*) quadrantic defects; (*d*) peripheral crescentic defects; and (*e*) no abnormal changes. When vision was failing a concentric

contraction of the visual fields was sometimes found added to the above variations.

(6) An attempt was made to group the symptoms and physical signs as focal, neighbourhood and those due to increased intracranial pressure. Focal symptoms and signs were limited to unformed visual hallucinations and distortions of the visual fields. Neighbourhood symptoms and signs were of considerable importance in localization, and with the exception of disturbances of speech were probably the result of œdema or vascular changes in the neighbourhood of the tumour, or of pressure upon adjacent structures, sometimes increased by raised supratentorial pressure. Symptoms and signs resulting from increased pressure in some cases were confusing and increased the risk of an error in localization.

(7) In the majority of cases symptoms of increased intracranial pressure dominated the clinical picture from the beginning; in a few epileptiform attacks occurred for some time; and in others there was a history of exacerbations of increased intracranial pressure for as long as two years.

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NERVOUS AND MENTAL PHENOMENA ASSOCIATED WITH PAROXYSMAL TACHYCARDIA.[1]

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Introduction.

It is my purpose here to call attention to the various nervous and mental manifestations which may be associated with paroxysmal tachycardia. Neurologists or psychiatrists are prone to consider tachycardia as belonging to the domain of the general physician, and as a consequence to overlook a syndrome that frequently has distinct nervous or mental phenomena associated with it. These phenomena may be so marked and may present such a variable picture as to mask the underlying cause, giving rise to many misconceptions and leading to incorrect diagnosis. The literature on paroxysmal tachycardia is voluminous, but comparatively little has been written regarding the nervous and mental aspects of the problem. As far as I know, no one has assembled the observations of physicians of the past who noted nervous or mental elements in the syndrome of paroxysmal tachycardia.

History.

Galen, in "De Pulsibus," mentioned sudden paroxysms of palpitation in young persons of apparently perfect health, not infrequently terminating fatally by syncope. In Wood's "Practice of Medicine," 1852, a most accurate description of paroxysmal tachycardia is given and the condition is recognized as being functional. Von Bamberger, in his "Lehrbuch," commented on the syndrome and mentioned the occurrence of headache, dizziness, scotoma, tremor, and even unconsciousness in the attacks.

From these early and somewhat vague references one may turn to the year 1867, when, within a brief period, four writers published reports of cases in the *British Medical Journal* on "Unusually Rapid Action of the Heart." In a case reported by Cotton, a patient had associated sensations of faintness, and in a case reported by Watson

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the patient died in his fourth attack. In spite of these reports, little of special interest appeared until 1874, when Huppert described the condition but stated incorrectly that previous reports had not appeared in the literature. Proebsting, in 1882, was more fortunate and, besides describing three cases of his own, was able to review reports of numerous cases from the literature. A few years later Bristowe made several pertinent observations. He noted that as far as the heart is concerned the condition is functional, and if any cardiac disease is present it is purely accidental.

It remained, however, for Bouveret, in 1889, to realize the significance of the syndrome, and he coined the term "tachycardie essentielle paroxystique." He published an exhaustive and masterly review of the subject and left comparatively little to add other than details, reports of unusual cases and the more recent and important electrocardiographic studies. He fully appreciated the significance of associated cerebral phenomena and attributed them to ischæmia or cerebral circulatory changes. It is strange that Bouveret's work made but slight impression at the time, and approximately ten years elapsed before any noteworthy contribution was published, although occasionally case records appeared under the heading of cardiac neurosis, cardiac epilepsy, and even under the heading of gastric neurosis.

Herringham, in 1897, reviewed reports of fifty-three cases gathered from the literature. He pointed out that there was no essential difference in the cases of organic cardiac disease, and that Bouveret was not justified in excluding such cases. He also recognized various cerebral manifestations and mentioned, in particular, one case from the literature in which the attack was initiated by loss of consciousness..

Schlesinger was the first to make a thorough study of paroxysmal tachycardia in relation to the central nervous system. He studied fifteen cases, and noted a great variety of neurological manifestations. He observed that in spite of marked tachycardia the patients usually were able to carry on their accustomed physical duties. He emphasized the fact that in the majority of cases careful clinical study did not reveal organic disease, and that, beyond the cardiac condition, evidence of neurosis could not be established. Unlike most observers, however, he noted that neurasthenia or hysteria might be associated, although such states were rather the results of the tachycardia than the cause. Schlesinger observed paroxysmal tachycardia in a patient subject to Quincke's œdema, and it was his belief that the œdema played a causative part in the production of the tachycardia. He also noted the associa-

tion of unconscious attacks with paroxysmal tachycardia, and reported four cases in which attacks of paroxysmal tachycardia were initiated by a convulsive attack and unconsciousness. In one of these cases, circumscribed œdema was always present with an attack and never occurred between attacks. He suggested that possibly the convulsive attacks in this case were due to œdema of the cortex, for not infrequently convulsive attacks are associated with circumscribed œdema. He was able to find, in the literature, reports of ten cases of paroxysmal tachycardia with convulsive seizures, and in the majority of these cases he assumed the convulsions to be due to cortical œdema, vascular spasms, arteriosclerosis, or some other cerebral factor. Although the term " cardiac epilepsy " has been employed, Schlesinger did not consider paroxysmal tachycardia as an epileptic equivalent. He stated, however, that the more one studies paroxysmal tachycardia, the more tenable the theory of an epileptic factor seems to be.

As might be anticipated, every writer on the subject had some favourite mechanism to propound as the cause of paroxysmal tachycardia, but the consensus of opinion regarded it as a neurosis. The occasional association of migraine prompted the idea of an underlying migraine or gouty diathesis. Thomas and Post suggested that paroxysmal tachycardia might be the cardiac manifestation of migraine.

It is impossible to review all the hypotheses applying to the causation of paroxysmal tachycardia. The very fact that there was no unanimity of opinion lends weight to the conclusion that much was left for the future.

With the advent of the electrocardiograph added light was thrown on paroxysmal tachycardia. In 1924, Willius and Barnes presented an exhaustive and careful study of the subject. They classified tachycardia into (1) simple (nodal, auricular, ventricular), (2) auricular fibrillation, and (3) auricular flutter. In a supplementary study Barnes called attention to various cerebral manifestations of the disorder and reported several interesting cases.

Definition.

Paroxysmal tachycardia may be defined as a condition which is characterized by the following features: (1) Sudden change in the normal cardiac mechanism, producing a rapid rate, varying between 120 and 250 beats a minute; (2) the patient may or may not be entirely conscious of the great degree of cardiac acceleration; (3) the attack generally covers a definite period of variable length, from minutes to

hours, or even, rarely, days; (4) the tachycardia not only begins abruptly but usually ends abruptly; and (5) cerebral phenomena may be associated with the attack and may tend to dominate the clinical picture.

Ætiology.

Paroxysmal tachycardia is slightly more prevalent in males than in females. It may occur at practically any age, ranging from 7 to 70 years. It is, however, comparatively rare in children. Willius found reports of only twelve authentic cases in childhood, and added one of his own, that of a girl aged 7. The greater number of patients present themselves between the fourth and sixth decades of life. As a rule the attacks continue over a period of many years. Hoffmann reported one case in which the attacks had existed for fifty years. I have seen several cases in which it had existed over a period of thirty years. As a rule the attacks vary considerably as to frequency. There may be recurring attacks for several months, or even years, and then there may be a free period of months or years. Again, a patient may have an attack once or twice a year, over a long period of time. Although previous diseases, such as tonsillitis, influenza and rheumatic fever are frequently recorded, it must be admitted that the percentage is hardly sufficient to warrant a positive deduction. Exciting causes are numerous and belong to the conditions usually associated with functional disorders, such as emotional disturbances, exertion (such as dancing and skating), ingestion of food, flatulence, sudden change in posture, excessive use of tobacco or alcohol, and prolonged pain; at times percussion over the heart will precipitate an attack. The attacks may, however, have no apparent exciting factor and may occur without warning and even during sleep.

Morbid Anatomy.

The outstanding factor in the pathology of paroxysmal tachycardia is the absence of organic changes. From very early times efforts have been made to single out some cardiac or extracardiac lesion which might be a factor in the production of the disease. Schlesinger reported a case in which at necropsy compression of the right vagus nerve was found. Since his observations several others have reported similar lesions, but when the evidence for a lesion of the vagus nerve, as a cause of tachycardia, is summed up, there is comparatively little basis for a conclusion. Reinhold, in 1906, reported a case in which there was syphilitic basal meningitis, and one in which there was a tumour of the left cerebellum, but he himself concluded that these lesions might be

wholly unrelated to the tachycardia. It is unquestionable that, in the presence of pathological changes in the heart, the condition takes on a more serious aspect, but pathological change in tissue is contributory, not primary. Summing up the evidence at hand, one must conclude that up to the present time pathological investigation has not revealed any single factor of positive value. Yater, in a study of the pathologic changes in arrythmia, studied 154 hearts without finding any specific histological lesion to account for the tachycardia.

Nervous and mental symptoms are probably secondary to interference with the normal cerebral circulation. Owing to the rapid beat of the heart, the ventricles fail to fill properly, and as a result the volume of the flow of blood to the brain is decreased, resulting in symptoms usually attributed to cerebral anæmia. The presence of arteriosclerosis naturally would serve as an added factor in producing circulatory changes during an attack, and may in part account for cerebral manifestations occurring more commonly in adults.

Symptoms.

The clinical picture of paroxysmal tachycardia is so characteristic and so fully described by its definition that it does not need detailed description. The important point from the standpoint of the physician is to keep the condition in mind, and in the presence of recurring, unexplained cardiac or cerebral phenomena to question closely concerning the exact symptoms. If need be, he should endeavour to witness an attack; also, if possible, he should obtain an electrocardiographic tracing during the attack. Lewis has pointed out that the less frequent the attacks the longer they last. In any given patient the duration of attacks is fairly constant, so that the individual paroxysms are similar.

As a rule the patient suddenly becomes aware of cardiac palpitation, the heart may seem to turn over or "flop over" and race off at a rapid rate. Often the attacks are of short duration, and the patient may continue with the usual work without especial embarrassment. Lassitude, exhaustion, sweating and even prostration may supervene. Throughout the attack the cardiac rate remains the same, and as a rule cessation is as abrupt as the onset, although gradual subsidence not infrequently occurs. Within a brief interval the patient regains his normal composure and status, although general lassitude may persist for several days. As a rule, the patient does not seek advice with a first attack. The tachycardia may go unnoticed for years and may be considered as some trouble not cardiac in origin.

Diagnosis.

The diagnosis of paroxysmal tachycardia is comparatively simple if the condition is kept in mind. The difficulty is that only too often the patient is not seriously impressed by the tachycardia but regards the cardiac activity as secondary to dizziness, faintness or some factor in the condition. On the other hand, many of these patients live in dread of recurring attacks, and as a result definite anxiety neurosis develops, so that one may have considerable trouble in appreciating the existence of two separate syndromes. Because of the variable associated phenomena, many of these cases are incorrectly diagnosed as vertiginous attacks, neurosis (cardiac, anxiety, gastric and menopause), *petit mal*, syncopal attacks, epilepsy, chronic nervous exhaustion and migraine-epilepsy complex.

Naturally, many of these cases are classified under the heading of cardiac neurosis. Hirschfelder has attempted to differentiate the purely neurotic type of case and the essential or myogenic type on the following basis: In the neurogenic type the tachycardia sets in gradually and subsides gradually, whereas in the myogenic type it sets in at a single leap and the heart-beat reaches its maximal rate within one or two cardiac cycles. Willius, in considering cardiac neurosis, stated that many cases of paroxysmal tachycardia are neurogenic in origin, and that paroxysmal nodal tachycardia is the type that is most frequently neurogenic. He has never observed paroxysmal auricular fibrillation, auricular flutter or ventricular tachycardia in hearts not affected by disease.

In some instances a differential diagnosis is not easy; thus, one patient may present gastric complaints, such as recurring attacks of flatulence associated with dizziness and vertigo. Closer examination reveals the fact that the condition is entirely cardiac but the patient has misinterpreted his own sensations. In any given case, if doubt exists, a final opinion should not be rendered without electrocardiographic study.

Barnes obtained electrocardiograms in thirteen cases during attacks; the origin of the tachycardia was as follows: in seven, nodal; in three, ventricular; in one, auricular; and in two, auricular flutter.

In patients who present syncopal attacks, or unconsciousness, it is easily seen that at times distinction between paroxysmal tachycardia and epilepsy is difficult. In paroxysmal tachycardia there is usually a warning and the patient frequently is able to prevent the attack by lying

down. If unconsciousness supervene the patient rarely falls, but tends to slide to the ground. There is absence of convulsive movements, although, in the excitement, a few movements of the patient may be recalled by the witnesses as convulsive acts. Sphincter disturbance is absent, and on recovery, even though there be exhaustion, stupor or sleep is absent. True convulsive seizures also may occur, but they are comparatively rare, and unless distinctly associated with an attack of tachycardia must be considered as an independent condition. As a rule, in epilepsy the patient becomes cyanotic, whereas in paroxysmal tachycardia he becomes pale. A distinguishing feature in paroxysmal tachycardia is the fact that after the attack has begun, change in posture does not affect the cardiac rate. As a rule, in physiological acceleration, as in exophthalmic goitre and infectious processes, the rate responds to rest and postural changes. Vomiting, prostration, or even delirium must be recognized as occasional phenomena associated with paroxysmal tachycardia. It should be kept in mind that persistent tachycardia, in which the cardiac rate is 140 or more, and in which this rate is maintained in a variety of circumstances, is almost always due to a new and pathological heart rhythm [15].

Nervous and Mental Phenomena.

The recorded frequency of nervous and mental symptoms in paroxysmal tachycardia depends on the investigator's attitude in including mild symptoms, such as anxiety, dizziness and vertigo, as part of the syndrome. In about 20 per cent. of cases of paroxysmal tachycardia in the Mayo Clinic, mental phenomena of varying degree are encountered. Definite nervous manifestations are somewhat less common; they occurred in the same group of patients in about 15 per cent. of the cases, the symptoms varying from mild dizziness to attacks of unconsciousness. In spite of the rather infrequent occurrence of mental disturbances, it is noteworthy that many patients are entirely free of associated psychotic disturbance and have learned to accept their attacks without any marked concern.

The more common disturbances referable to the nervous system may be grouped as nervous and as mental phenomena. Nervous phenomena are dizziness, vertigo, syncopal attacks, visual disturbances such as temporary blindness and scotoma, attacks of unconsciousness, convulsive seizures, paræsthesia, pain and transitory aphasia. Among the more common mental phenomena associated with the attacks of paroxysmal

tachycardia are anxiety, depression, agitation, mental and physical exhaustion, rarely mild delirium, and at times hysterical manifestations.

These associated phenomena are not confined to any special age. They appear more often in cases in which the origin of the stimulus and the propagation over the heart shows the greatest deviation from normal. Thus, Barnes found that cerebral manifestations were present in 60 per cent. of cases of paroxysmal tachycardia in which the impulse originated in the ventricle; in 41 per cent. of the cases there was nodal tachycardia; in 25 per cent. auricular tachycardia, and in 6 per cent. auricular flutter. As far as can be determined, the occurrence of cerebral manifestations does not affect the prognosis in paroxysmal tachycardia.

The exact relationship of nervous or mental symptoms to the attack of tachycardia is extremely variable. Occasionally the attacks are ushered in by some symptom such as depression, anxiety, vertigo, or even unconsciousness, so that the entire attention of the patient is focused on the initial symptom and the underlying condition is not recognized and in itself causes no concern.

Frequently patients with paroxysmal tachycardia are treated for nervousness, and for years the condition may be classified under the heading of cardiac neurosis, anxiety neurosis, chronic nervous exhaustion, menopausal neurosis, and so forth. In any given case, if there are any associated disturbances with the attack they tend to remain more or less constant. Thus, a patient with fainting attacks will continue with variable degrees of syncope, and a patient with paræsthesia will continue to have paræsthesia.

That psychic factors may play an important part in the causation of attacks is undoubtedly true. Kuré, in a study of the psychic manifestations, was able to show that psychic influences, such as working out mathematical problems under strain and stress, might precipitate an attack. It is because of this frequent psychic association that in many cases the diagnosis of anxiety neurosis is made.

Prognosis and Treatment.

Barnes, in a previous study, pointed out that the prognosis in cases of paroxysmal tachycardia, without signs of organic cardiac disease, is good, and that the attacks are compatible with long life; but if attacks occur in conjunction with organic cardiac disease, the prognosis is determined by the type and degree of the underlying cardiac injury. Since,

in the majority of instances, there is no pathological change in the heart, one is justified in assuming a favourable prognosis as far as life is concerned. The duration of the disease is extremely variable. At times a few isolated attacks conclude the picture. More often the condition tends to recur over many years; the average duration, at the time the study was made, was about ten years. I have seen several cases which have extended for more than thirty years, and some writers have reported cases of fifty years' duration. That the condition must not be treated lightly is evident from the fact that I have seen death occur as a result of a paroxysm in a case in which no cardiac or other pathological cause of the condition could be found. One woman, aged 23, died during an attack, and at necropsy chronic fibrinous myocarditis, dilatation of the left ventricle, a defect of the intraventricular septum, and an aneurysm were found. There is nothing in the association of nervous or mental phenomena which alters the prognosis. However, nervous and mental changes are more prone to occur in those cases in which there is a definite cardiac lesion. Attacks of paroxysmal tachycardia also may prove serious or even fatal if associated with pulmonary disease. I have had one patient who died in the course of post-operative bronchopneumonia, but death occurred as an immediate sequel of an attack of tachycardia.

Of paramount importance in the treatment of paroxysmal tachycardia are the recognition and treatment of any pathological change in the heart. Whether or not there be a cardiac lesion, the patient must fully appreciate the nature of his condition, for there is little doubt that emotional strains and stresses do precipitate attacks. The elimination of toxic agents, such as alcohol and tobacco, is important, for not infrequently the reduction of smoking alone may lessen the frequency of the attacks. With the onset of the attack complete rest and relaxation may shorten or may even abort an attack. Vagal pressure, or the production of vomiting, may abort an attack; patients frequently learn to induce vomiting to relieve the condition. In several instances the removal of foci of infection, such as the tonsils, has seemingly been of value. Morphine sulphate rarely does much good unless it produces vomiting, and naturally, in recurring attacks, its use is contra-indicated. Digitalis is of no value. Quinidine sulphate given in doses of 3 to 4 gr. (0·20 to 0·24 grm.) every three or four hours during the day and night appears to be the most helpful remedy, as it tends to depress the irritability of the auricle. If the attacks are frequent, the quinidine may be given in doses of 3 gr. (0·20 grm.) three to four times a day over an indefinite

period of time. Finally, all emotional strains and stresses should be eliminated from the life of the patient.

Cases Illustrating the More Common Nervous and Mental Phenomena.

Case 1. *Paroxysmal nodal tachycardia with vertigo.*—A man, aged 62 years, had been subject to severe attacks of vertigo for the last four years. He recalled, only after direct questioning, that these attacks had been attended by sudden seizures of tachycardia. The Wassermann reaction of the blood was positive, and signs of syphilitic aortitis were present. A radial pulse could not be obtained in the right arm, but collateral circulation was well established. There was marked arteriosclerosis. After the patient had been under observation a few days severe vertigo developed. The pulse-rate was 160, and an electrocardiogram taken during the attack identified the paroxysm as nodal tachycardia with a pulse-rate interval of zero and a cardiac rate of 150. The vertigo ceased when the heart resumed its normal rate.

Dizziness or vertigo is one of the most common associated nervous manifestations encountered in paroxysmal tachycardia. This case is selected because of the electrocardiogram. As far as could be determined, syphilis did not play a part in the causation of the attacks. The patient has now been under observation for five years, and at the age of 67 remains in fair health and only rarely has an attack of tachycardia.

Case 2. *Paroxysmal tachycardia with syncope and loss of consciousness.*—A nurse, aged 22, five years before she came to the clinic had begun to have what she considered to be fainting attacks. At first they occurred infrequently, but for fourteen months had been coming on about every six weeks. A diagnosis of epilepsy had been made. The attacks usually had come on while the patient was working hard or was under strain. Suddenly a peculiar sensation would pass over her, the heart seemed to stop for a moment and then beat rapidly. With this a feeling of weakness and faintness came on, and unless she could lie down or get fresh air, she would sink to the floor and might be unconscious for a moment, or as long as fifteen minutes. She was observed in one attack; the rate of the heart was about 200 for about an hour and a half; then, suddenly, it returned to normal. Following an attack there was a sense of exhaustion lasting for several hours.

In this case the dominant feature was the loss of consciousness. The result was an erroneous diagnosis of epilepsy.

Case 3. *Paroxysmal tachycardia with convulsions.*—A woman, aged 50, had begun to have attacks of unconsciousness thirty years before she came to the clinic. The attacks recurred several times a year. Suddenly she would feel dizzy and weak, and, unless she could get to a chair, would fall unconscious.

She never had had any convulsive movements, but she sometimes became rigid. Attacks occurred at night, and she awoke with the onset. The attacks always began with sudden onset of rapid heart-rate, a cold sensation passed about the mouth, and the lips tingled, objects grew dark before her and syncope followed. Attacks lasted from a few minutes to seven hours.

In this case a diagnosis of epilepsy had been accepted for a long time, and it was only on very close questioning that a history of cardiac abnormality was obtained, as all previous interest had centred about the convulsions. In spite of the long history, the attacks are now less severe and there is no indication of mental deterioration.

Case 4. *Paroxysmal tachycardia associated with syncope and unconsciousness.*—A clergyman, aged 43, twenty years before he came to the clinic had begun to have attacks of tachycardia lasting about a half hour. Usually the attacks had been brought on by exertion, but also occurred without apparent cause. Seventeen months before he presented himself for examination, he suddenly had had an attack while preaching, had become weak, had fallen and had been unconscious for five minutes. Seven months later a similar attack had occurred. In the last five months he had had four attacks. The attacks were usually preceded by a depressed feeling, discomfort in the epigastrium, and then the sudden appearance of tachycardia. If he could sit or lie down he could usually ward off the attack. If he were unable to ward off the attack, the heart-rate continued at about 200 beats a minute until he sank to the floor and remained unconscious for five to ten minutes. Following an attack he was weak, but not sleepy. He experienced some anxiety during the attack, but anxiety did not precipitate an attack.

The first impression was that the cause of the condition was epilepsy of the *petit mal* and *grand mal* types. The patient also was rather markedly anxious and depressed as the result of these attacks, but as far as could be determined the anxiety did not precipitate attacks.

Case 5. *Paroxysmal tachycardia with anginoid type of pain.*—At the age of 17, following influenza, a woman began to have infrequent attacks of tachycardia. At the age of 45 the attacks had become more severe, lasting from two to thirty-six hours. Since the onset of the tachycardia she had been subject to pain running down the left arm, especially in the region of the deltoid muscle and at the elbow (not ulnar distribution). On a few occasions she had had contraction of the hands and feet, simulating tetany. During an attack she had a heart-rate of 200. An electrocardiogram taken during an attack showed premature T-waves in leads I and II, and inverted T-waves in lead III. The Q.R.S. interval was at the upper limit of normal.

In this case the possibility of true organic disorder was naturally the first consideration. The pain, however, was not typical of true angina,

and when considered as part of the paroxysmal tachycardia, the syndrome justified an entirely different prognosis from that of severe organic cardiac disease.

Case 6. *Paroxysmal tachycardia with visual disturbance and symptomatic depression.*—In 1905, when the patient, a man then 38, was at work, suddenly everything grew dark before his eyes and he had a sensation as of "stars falling" before his eyes. He slowly fell forward, but saved himself and sat down for several minutes. He had been aware of marked tachycardia and dyspnœa. In 1915 he had had a similar attack that had been followed by weakness and depression for several days. A physician had told the patient at this time that his heart was "very bad," and that he would "fall over some time and forget to wake up." No further attacks had occurred until 1919, when the trouble had returned. In this attack at this time he sank to the ground, but was not convulsed. Since then up to the time of a visit in May, 1928, he had had, on an average, three or four attacks a year, lasting from three to ten minutes each. Following attacks he had been depressed and had dreaded not being able to do his work, everything had seemed wrong, words had confused him and he had been unable to do his usual work. He had had similar depression without attacks of tachycardia, but invariably had become despondent with an attack. Examination had been entirely negative in 1921 and was negative again in 1928.

Visual disturbances are not uncommon in paroxysmal tachycardia and may vary from momentary blankness to marked scotoma. In this case the depression may have been provoked by an unwise remark on the part of the physician.

Case 7. *Paroxysmal ventricular tachycardia with blindness.*—A man, aged 34, complained of attacks of vertigo and blindness, followed by rapid action of the heart. The attacks had begun two months before his visit to the clinic. The patient used tobacco and coffee to excess. He suffered from migraine, but there was no relation between the headaches and the attacks of rapid action of the heart. In a typical attack he first became totally blind and dizzy for a few seconds, after which "the heart ran away with itself." He felt weak during the attack, was pale and bathed in cold perspiration. The attacks lasted about four hours and ceased after he had vomited. The electrocardiogram taken during an attack gave evidence of ventricular tachycardia with a rate of 257. General examination gave negative results, except for infected tonsils, which were removed. Three years later the patient was free from attacks, and from symptoms of myocardial failure.

The possibility of a migraine equivalent was considered but could not be established. The removal of the infected tonsils and limitation of indulgence in tobacco and coffee may have been factors in improvement.

Case 8. *Paroxysmal tachycardia, with paræsthesia and aphasia.*—A woman, aged 33, had had enlargement of the thyroid gland since the age of 17. Since influenza in 1919, she had not been well and had been nervous and easily exhausted. About a year before she came to the clinic she had begun to have attacks which always had started with sudden onset of rapid action of the heart. Prickling and numbness about the mouth and of the hands and feet followed an attack. For five to fifteen minutes she felt helpless and unable to talk. Following the attacks she was exhausted. The attacks were brought on by worry, nervous strain, or quarrels.

Paræsthesia is a rather common manifestation of paroxysmal tachycardia and may be of varying degree.

Case 9. *Paroxysmal tachycardia; nocturnal attacks; syncope associated with unconsciousness.*—A man, aged 38, five years previous to his visit to the clinic had begun to have sudden attacks characterized by rapid beating of the heart, paræsthesia, and twitching of the left arm. The attacks occurred about monthly, and lasted from three to ten minutes. On two occasions they had been associated with loss of consciousness. The attacks occurred at night, waking him out of a troubled sleep. In the convulsive attack his limbs were contracted, but clonic movements had never been present. A diagnosis of epilepsy had been made.

The occurrence of attacks at night is not common; it need not be interpreted as a sign of epilepsy. The character of the convulsions was not epileptic, and there were no features in the case to suggest a localized cerebral lesion.

Summary.

The syndrome of paroxysmal tachycardia is one that has long been recognized by general physicians, but that has been given little consideration by neurologists and psychiatrists. There seems little doubt that nervous and mental phenomena are frequently associated symptoms and that they may so dominate the picture as to lead to incorrect diagnoses. Although emotional elements may play a part in the syndrome they may be entirely absent, and the symptoms are then directly the result of a cerebral vascular change. It is therefore advisable, in the presence of any recurring paroxysmal type of attack, to bear in mind the possibility of paroxysmal tachycardia and its associated phenomena.

Conclusions.

(1) Paroxysmal tachycardia is of fairly common occurrence, and because of its associated nervous and mental manifestations merits the consideration of neurologists and psychiatrists.

(2) Its chief characteristic is its abrupt onset and cessation.

(3) It is more common in neuropathic persons, and there may be other evidence of functional nervous disturbances.

(4) The presence of organic pathological changes in the heart need not modify the clinical picture, although the prognosis varies with the degree of cardiac injury.

(5) The nervous and mental phenomena may vary from slight anxiety or dizziness to delirium or convulsions.

(6) Although it is not possible to establish either an isolated ætiological factor or a precise pathological lesion, paroxysmal tachycardia represents a clinical entity and should be so recognized.

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Notices of Recent Publications.

Encephalitis im Kindesalters. By A. Eckstein. S. 172. Berlin: Julius Springer. 1929. Price M. 12.

In view of the interest evoked to-day by the acute cerebral complications of the exanthemata and by other types of encephalitis, the appearance of Dr. Eckstein's monograph is particularly opportune. The author discusses fully the clinico-pathological aspects of that broad group of cases occurring in childhood in which acute cerebral manifestations occupy the foreground of the clinical picture, whether complicating some systemic disease or arising spontaneously. Naturally a large proportion of the monograph is concerned with the symptomatology of acute and chronic epidemic encephalitis. Epidemic forms of encephalitis, other than encephalitis lethargica, are next considered. Within this category, Eckstein includes the cases of "acute serous encephalitis," reported from Boston and New York by Brown and Symmers; the Japanese epidemic described by Kaneko and Aoki; the cases of Stoos; and lastly, the "X disease" of Australia. Thereafter, the sporadic cases of encephalitis in childhood are described, illustrated by a long series of the author's personal cases. "Para-infectious" encephalitis is considered in some detail, followed by a careful study of post-vaccinal encephalitis. A useful table is given, setting out the particulars of ninety-three examples reported in Germany, including three patients from the author's series. Altogether, this monograph constitutes an able and readable presentation of the subject, and is printed and illustrated in the excellent manner which characterizes Springer's publications.

Tumeurs médullaires associées à un Processus syringomyélique. Par N. Jonesco-Sisesti. P. 295. Paris: Masson et Cie. 1929. Price 45 frs.

The rare but interesting condition in which an intramedullary tumour of the spinal cord is associated with syringomyelic lesions above and below the level of the growth is carefully studied from both its clinical and anatomical aspects in this monograph. The author had the opportunity of observing several cases under Professor Guillain, and he has also collected many others from the literature. From an analysis of these cases he concludes that sensory symptoms generally appear first and are usually the most prominent throughout the course of the illness. The earlier disturbances are generally radicular in distribution, but later they are more widespread. For a time the characteristic syringomyelic dissociation of sensation is found, but tactile sensibility diminishes rapidly. The motor symptoms are less characteristic, and, as the sensory, tend to vary from time to time.

The author emphasizes the importance of an examination of the cerebrospinal fluid, for though the typical Froin syndrome is usually present the cerebrospinal pressure is not raised. The primary tumours are classified as fibrogliomata, ependymogliomata, and complex tumours, chiefly teratomata, while the syringomyelic lesions are attributed to a gliomatosis which originally surrounds the tumour but later spreads throughout the cord.

Spezielle Histopathologie des Grosshirns. Von Professor Dr. A. JAKOB. Erster Teil, mit 266 zum Teile farbigen Abbildungen im Texte. S. 412. Leipzig und Wien: Franz Deuticke. 1929.

This valuable addition to Aschaffenburg's "Handbuch der Psychiatrie" deals with the special histopathology of the brain, and is as trustworthy and informative as its author's reputation would lead one to expect.

After an introduction in which the problems and limitations of research in this field are discussed in a temperate and comprehensive way, there is a section on tuberculosis of the brain and its membranes. The body of the book is devoted to syphilis, considered under five heads: syphilitic meningitis and meningoencephalitis, vascular lues of the brain, cerebral changes in congenital syphilis, general paralysis of the insane, and the "tabes-psychoses." The last hundred pages of the volume deal with trypanosomiasis, disseminated sclerosis, and diffuse sclerosis (encephalitis periaxialis diffusa). The remaining disorders of the brain, including extrapyramidal conditions, are to be considered in the forthcoming second part.

The relationship of anatomical changes to clinical features is not overlooked, nor the necessity for suspending judgment on many points, where, however, the conflicting views are presented and summed up with great lucidity; this is done, for example, in a masterly way in the "observations on the pathogenesis of general paralysis."

The references to literature are full, and the photomicrographs numerous and clear.

Ueber einige neuere diagnostische und therapeutische Verfahren in der Neurologie. Von E. VON THURZO. S. 78. Berlin: S. Karger.

A series of addresses, mainly on diagnostic and therapeutic problems, which the author has given before different societies are brought together in this small volume. The more important are those on the abduction reflex of the toes, on the introduction of air by the lumbar and suboccipital routes for the purpose of encephalography, and on the modification of the Lange reaction introduced by Sicard and Haguenau.

The article on the influence of malarial infection in the early stages of syphilis on the subsequent development of neurosyphilis brings out some interesting points. Apparently the infection does not make the occurrence of nervous symptoms less likely, but when either tabes or dementia paralytica develops it runs a more favourable course.

The Pathology of the Eye. By JONAS FRIEDENWALD, A.M., M.D., F.A.C.S. Pp. 346, with 253 illustrations. London: Henry Kimpton. Price 21s. net.

In this book an attempt is made to link the pathology of the eye to that of the other tissues of the body. This attempt is only partially successful, for one cannot expect tissues so specialized and delicate as those of the eye to react in the same way to bacterial and metabolic poisons as less highly differentiated tissues. In this way the problems of eye pathology are similar to that of neuropathology, but while the eye is more limited in size it contains a greater variety of special tissues than the brain. Several of these, such as the cornea, the lens and the vitreous, are avascular and are therefore more vulnerable than any vascularized tissues. The author has indeed, as far as knowledge goes, given a very clear picture of ocular pathology, linking it with general pathology on the one hand and with what can be seen during life on the other hand. He is disinclined to discuss theories of causation unless these are well established, and prefers a confession of ignorance or of philosophic doubt to a presentation of the theories which have at one time or another been put forward to explain phenomena. While this attitude saves space, it is in some respects disappointing to the neurologist, especially in relation to the causation of papilloedema and optic neuritis and atrophy. The various problems related to glaucoma are rather more fully discussed, but the work of Duke Elder on the circulation of the intra-ocular fluids is scarcely mentioned. The parallelism between this circulation and that of the cerebrospinal fluid is close that the author might have benefited by a study of the recent work on the latter subject. The book is richly illustrated with photomicrographs, most of which are excellent.

The Meaning of Sacrifice. By R. MONEY-KYRLE. (International Psycho-Analytical Library, No. 16.) Pp. 273. London: Hogarth Press and Institute of Psycho-analysis. 1930. Price 18s.

Since the publication of Freud's "Totem and Tabu" a number of writers have applied psycho-analytic theories to the study of anthropology. Dr. Money-Kyrle has here collected from standard works the available data about rites of sacrifice and attempted to elucidate their meaning, expressed in terms of unconscious impulses. To these unconscious impulses, as conceived by the Freudian School, and particularly to the significance and consequences of the Œdipus situation, he devotes the first part of the book. It is impossible to examine his conclusions and methods in a brief notice. The book is lucid and well-informed, less dogmatic than some similar works, and can be recommended to the attention of all who are interested in the interpretation of primitive customs.

Geschehnis und Erlebnis. Zugleich eine historiologische Deutung des psychischen Traumas und der Rentenneurose. Von ERWIN STRAUS. S. 129. Berlin: Springer. 1930. Price Rm. 6.60.

This little book represents an admirable attempt to clear up fog. Among nebulous terms, "psychic trauma" is one of the commonest used in psychiatry; Dr. Straus clarifies it on grounds of theoretical psychology and empirical observation of cases, using the familiar picture of compensation-neurosis as the means to this end. Time-relationships, with which he has concerned himself in an earlier paper, are regarded as the central problem around which are grouped related matters, such as the author's concept of "Deformation" as a fundamental feature in the psychopathology of neuroses and perversions.

The book is critical, close-woven and predominantly psychological.

Lebensschicksal und Persönlichkeit ehemaliger Fürsorgezöglinge. Von ADELHEID FUCHS-KAMP. S. 172. Berlin: J. Springer. 1929. Price M. 18.60.

This sociological study is the latest of the series of Heidelberg monographs on criminal psychology, in which, among others, Kurt Schneider's study of prostitutes and Homburger's of the criminal insane have appeared. The histories of sixty-five former inmates of the Flehingen Reformatory in Baden have been compiled from institution and Court records, letters, &c., and an examination of their present personality and social relations carried out with as much completeness as possible. From the data thus obtained interesting conclusions are drawn as to the relation of criminality to psychopathic disposition. The authoress makes no reference to the thorough and more extensive studies of this kind that have been made in the United States.

Handbuch der Geisteskrankheiten. Herausgegeben von OSWALD BUMKE. 2ter Band, allgemeiner Teil 2. Störungen des Wollens, Handelns, Sprechens. S. 377. Berlin: J. Springer. 1928. Price Rm. 37.70.

This volume covers the difficult field of disorders of speech and conduct. Professor Bostroem writes in the opening section a concise account of the psychology and pathology of the will: he emphazises, however, that any isolated consideration of the phenomena of will represents an abstraction.

K. Birnbaum is responsible for the section dealing with the psycho-reactive symptom formations. The difficulties in the way of delimiting such a group are discussed, and the general description—"psychopathological phenomena which appear as pathologically conditioned reactions to psychic stimuli"—is expanded with the lucidity and grasp one expects of this author.

Catatonic disorders are discussed in detail by Bostroem, who also deals with syndromes dependent on lesions of the basal ganglia.

A monograph of 120 pages on "Aphasia, Apraxia, Agnosia," by Thiele, of Berlin, concludes the volume. Considering the enormous literature on the subject and its peculiar difficulties one welcomes a careful review as this of the development and present state of opinion; the author has wisely contented himself with description and appraisal, a sufficiently onerous task, and the result is a well-proportioned compendium which will be of the utmost value to anyone concerned with the problem of aphasia and allied disorders.

Lehrbuch der Geisteskrankheiten. Von OSWALD BUMKE. Dritte Auflage. S. 806 mit 160 Abbildungen. München: Bergmann. 1929.

This well-known textbook by the Professor of Psychiatry in Munich has been cut down to a handy size and otherwise revised for the purposes of a third edition. Conservative and critical, it is a sound exposition of psychiatric teaching, embodying much personal experience and the results of research in Germany so far as the latter has been kept free from any tincture of the Freudian doctrine, with which Professor Bumke will hold no terms. It is a pity that the sections on treatment are relatively so brief; it is a sign of honesty, no doubt, but more discouraging than the present state of affairs would warrant, especially in the case of schizophrenia.

Manuel élémentaire de Psychiatrie. Par M. NATHAN. Pp. 319. Paris: Masson. 1930. Price 30 francs.

This little book is so unpretentious in its title that one can hardly find fault with its defects of omission, but there are other faults, of arrangement and nosology, which spring from good intentions, but are especially unsuited to an elementary manual. The author applies the modern views about syndromes and dynamic types of reaction with such zeal that the overlapping between his "psychoses" and his "psychiatric syndromes" is bewildering, while for the beginner the lack of any introductory sections on general psychopathology and symptomatology is another stumbling-block. Little attention is devoted to the work of any but French psychiatrists.

Insomnia. By H. CRICHTON-MILLER. Op. 172. London: Arnold and Co. 1930. Price 10s. 6d.

Its sub-title, "An Outline for the Practitioner," describes the scope of this book. Several pages are devoted to the nature of sleep and to its physical and mental concomitants, but owing to the absence of a critical survey of the numerous theories presented, the reader not familiar with recent investigations on the subject is in danger of being confused.

The psychological aspect of sleep and the psycho-therapeutical treatment of insomnia claim most attention, but the uses of physical and medicinal treatment are adequately considered.

La Folie et la Guerre de 1914-1918. A. Rodiet et A. Fribourg-Blanc. With 10 plates. Pp. 187. Paris: Félix Alcan. 1930. Price 30 francs.

Although this monograph concerns the cases of mental disorder met with in the ex-Service man of France, it is actually a textbook of psychiatry in itself and covers the whole ground of mental disorder. The authors come to the same conclusion as have English observers in the same field, that is, that while the war has coloured the form and substance of the psychopathic states, the conditions themselves were clinically exactly like those in civil practice, and, in addition, that the war has not brought into being a new form of mental disorder. What the war has certainly brought out, in the view of the authors, has been the germination of constitutional and hereditary peculiarities until these became, in the stress of warfare and its call upon the instincts, fully-fledged psychopathic states. As regards alcoholism, they again corroborate the views of English observers that the drug itself was not the cause of the disorders arising, but that the alcohol acted upon excitable tissue because of the circumstances of war and the constitution of the subject, producing chronic mental disorder long after the effects of the alcohol had worn off. One unusual feature of this volume is the really artistic plates, many of them excellent drawings of cases of mental disorder. The photographs, too, of schizophrenic attitudes are quite unique.

Körperbau und Geisteskrankheit. By Max Schmidt. Pp. 206. Berlin: Julius Springer. Price 16 marks.

The investigations of Kretschmer into the relationship of physique to mental reaction type has led to as rich a crop of investigations as any discovery in the last forty years of psychiatry, with the exclusion, perhaps, of Freud and his school. This work has been almost entirely confined to Germany, and this volume is another example of the extraordinary painstaking way of the German psychiatrists, and also, let it be added, of the public support that is given to the publication of these magnificent monographs. The monograph is a frankly statistical clinical study of the two great physical types, the pyknic associated with the cyclothymic disposition, and the asthenic (leptosome) physique associated with the schizothymic disposition. Only students of psychopathology who have a first-hand knowledge of this specialized department will be able to read this volume critically, but all psychiatrists should make themselves acquainted with what is a very important and striking contribution to the psychophysics of mental disorder.

The study of morphology alone is not likely to solve the problems of reaction type, but viewed in the light of ethnology on the one hand and metabolism on the other, these studies in morphology, or "Körperbau," may prove to be important landmarks on the psycho-pathological road. Those who are doing work of this character will be very grateful to the authors for their thorough and painstaking researches.

Psychiatrie du Médecin-Praticien. By M. DIDE et P. GIRAUD. Second edition. Pp. 453. Paris: Masson et Cie. 1929. Price 45 francs.

This textbook on psychiatry is constructed on very formal lines, and, on the whole, is not controversial in matter, although the authors do not take up a modern psychological point of view; that is to say, they are almost untouched by Freud, and in their consideration of the large group schizophrenia, they are inclined to differ rather strongly from Bleuler. There is, as is so commonly found in French textbooks, a somewhat bewildering subdivision of subjects. This is perhaps the result of what is undoubtedly the French genius for stylistic descriptions of symptoms and behaviour.

The book is very beautifully illustrated with photographs of persons in typical attitudes of mental disorder; some of them are certainly the best we have seen published in psychiatric textbooks. These pictures, in a way, are the keynote to the author's attitude. Although a book of only 450 smallish pages, there is an enormous amount of material, and for students especially engaged in this branch of medicine they will be sure to find many new clinical facts.

Innere Sekretion und psychische Prozesse. By W. N. SPERANSKI. Pp. 150. Berlin: S. KARGER. 1929. Price M. 8.

Dr. Speranski's monograph is a systematic attempt to organize our present knowledge of endocrinology and its relation to psychology. As a member of the Russian school, he naturally opens his thesis with a discussion of Hormono-reflexology, i.e., he relates the activity of the hormones to the total activity of the organism considered behaviouristically. The James-Lange theory of the emotions comes under examination in this connection.

The burden of the monograph rests mainly upon a physiological and somewhat philosophical discussion of the neuropathology of hysteria and katatonia. The subject that Dr. Speranski has chosen is one of the first importance to all students of neurology who are struck by the common mechanisms that seem to be underlying such clinical conditions as hysteria, catatonia, mutism rigidity and katalepsy.

To have attempted further to relate such neurologically functional states to the internal secretion is an heroic effort. The author has not succeeded on the clinical level, but he has made a contribution to the general problem. His thesis, however, illustrates the danger of interpreting all behaviour in terms of neural mechanisms that are still highly speculative. All attempts to subsume the bewildering variety of psychic phenomena under simple natural concepts are in danger of losing those physiological events which alone give meaning to psychopathological states.

Le Développement mental et l'Intelligence. Par HENRI PIÉRON. Pp. 95. Paris: Felix Alcan. 1929. Price 10 francs.

With his characteristic gift of perfect condensation, Professor Piéron has put into these four lectures all the current matter relative to mental testing as a measure of intelligence and its development.

In the first lecture on stages of mental development, the author indicates very emphatically the importance of correlating successive stages of mental maturing with physical maturing. He also stresses the importance of realizing the fact that socialization is a measure of full linguistic development.

Lecture II concerns the history and method of mental testing. Than this chapter of twenty pages we have never seen a more masterly survey of what is an immense literature. Most interesting lecture of all, particularly to the clinical psychologist, is that which discusses mental levels with their relation to "mental profiles."

No practical picture of a mind can be of clinical value unless it presents the differential endowment of a subject. The illustration cases of profiles at different mental ages are of great value. The last lecture is methodological and gathers up all the data of the preceding lectures in a plea for analytic evaluation of data. A small book by a master hand.

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To those who are not members of the Neurological Section of the Royal Society of Medicine the price is 8s. 6d. net, and the volume may be obtained through any bookseller.

EDITOR.